

**PROCESSING SPEED AMONG ADULT STROKE SURVIVORS WITH LEFT-  
HEMISPHERE DAMAGE WITH AND WITHOUT APHASIA AND NORMAL  
HEALTHY CONTROLS**

by

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Submitted to the Graduate Faculty of  
The School of Health and Rehabilitation Sciences in partial fulfillment  
of the requirements for the degree of  
Doctor of Philosophy

University of Pittsburgh

2017

UNIVERSITY OF PITTSBURGH  
THE SCHOOL OF HEALTH AND REHABILITATION SCIENCES

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# **PROCESSING SPEED AMONG ADULT STROKE SURVIVORS WITH LEFT-HEMISPHERE DAMAGE WITH AND WITHOUT APHASIA AND NORMAL HEALTHY CONTROLS**

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University of Pittsburgh, 2017

The primary goal of this study was to explore whether slow processing speed in people with aphasia (PWA) is specific to this language disordered population. Based on the two different perspectives of general slowing and reduced processing speed, the information processing speed was explored using reaction time (RT) data.

The following specific issues were investigated: 1) Whether observed significant time differences are aphasia-specific, based on the comparisons between people with left hemisphere damage without aphasia (LHD) and PWA resulting from left hemisphere damage; 2) whether observed significant time differences are evident in both the nonlinguistic (CRTT-RT1-3 assessing motor speed, simple RT and movement control speed) and linguistic (letter comparison tasks 1 & 2 and lexical decision time) domains, compared between normal healthy controls (NHC) and PWA. The following experimental questions were investigated in this study:

1. Are there significant differences in the average RT per item among the average of the three identified nonlinguistic tasks and the average of the three identified linguistic tasks among the PWA, LHD and NHC groups?
2. Are there significant RT differences between PWA and NHC, between PWA and LHD, and between LHD and NHC for each task?

Generalized Linear Mixed Effects Models and Brinley plots, using regression analyses, were used to compare the magnitude of RT differences among groups and tasks.

**RESULTS:** The results of the mixed effects model revealed significant main effects for groups and domains, and no significant interactions among groups or domains. The two brain-

damaged groups (PWA and LHD) produced significantly longer reaction times across tasks than the NHC group. The PWA groups' reaction times were significantly longer than the LHD group across simple perceptual and more cognitively complex tasks except for the CRTT-RT1-3 sensory-motor tasks.

**CONCLUSION:** Aphasia-specific slowing, as well as left-hemisphere damage-related slowing was demonstrated as evidence by significant differences between the two brain-damaged groups and the NHC group. Therefore, the observed slowing in the PWA group appears to be due to both aphasia-specific and brain-damaged related slowing. Domain-specificity was not observed as significant slowing occurred in both linguistic and nonlinguistic tasks in both brain-damaged groups.

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## LIST OF ABBREVIATIONS

ABCD = Assessment Battery of Communication in Dementia (Bayles, Kathryn, Tomoeda, & Cheryl, 1993)

AIC = Akaike's Information Criterion

BDAE = Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983)

CMLP = Cross modal lexical priming

CRTT = Computerized Revised Token Test (McNeil, Pratt, Szuminsky et al., 2015).

CRTT-RT1-3 = Computerized Revised Token Test Reaction Time Battery Tasks 1 through 3.

CRTT-RT1 = Computerized Revised Token Test Reaction Time Battery Tasks 1

CRTT-RT2 = Computerized Revised Token Test Reaction Time Battery Tasks 2

CRTT-RT3 = Computerized Revised Token Test Reaction Time Battery Tasks 3

DLA = Delayed lexical access hypothesis

LHD = Left hemisphere damaged people without aphasia

NHC = Normal healthy controls

PICA = Porch Index of Communicative Ability (Porch, 2001)

PWA = People with aphasia

RSVP = Rapid serial visual paradigm

RT-a = Reaction time

RT-b = Response time

RT-ab = Reaction time (RT-a) and Response Time (RT-b)

SBC = Schwarz's Bayesian Criterion

SOA = Stimulus onset asynchrony

SOAP = Subject-relative, Object-relative, Active, and Passive (Love & Oster, 2002)

SOP = Speed of processing

SRT = Simple reaction time

SDT = Stimulus detection time

STM = Short-term memory

WAB = Western Aphasia Battery (Kertesz, 1979).

WM = Working memory

## **1.0 INTRODUCTION**

There is evidence, though controversial, supporting the notion that people with aphasia (PWA) are slow in their language performance. The evidence is not robust and the mechanisms of the slowness are unclear. For instance, Hochstenbach, Mulder, van Limbeek, Donders, & Schoonderwaldt (1998) reported that more than 70% of 229 aphasics exhibited slow information processing on an extensive neuropsychological test battery. PWA have also been reported to be slow, even after they are reported to have fully recovered (Neto & Santos, 2012).

Slowed language performance evidenced by longer response/reaction times and reading times has been attributed to delayed stages of processing in building representations, caused by failed computations or by limited or poorly deployed language-related cognitive resources. In order to more precisely account for the slow processing performance that occurs in PWA, some researchers have argued that they have slowed syntactic or lexical activation (e.g., Haarmann & Kolk, 1991; Love, Swinney, Walenski, & Zurif, 2008; Ferrill, Love, Walenski, & Shapiro, 2012; Piñango, Zurif, & Jackendoff, 1999; Piñango, 2000, Burkhardt, Piñango, & Wong, 2003). Others have hypothesized that the slowness is attributed to the inefficient allocation of cognitive resources required for computing syntactic structures, maintaining information in working memory, activation and inhibition of linguistic units, and deriving the meaning of a sentence (McNeil & Kimelman, 1986; McNeil, Odell, & Tseng, 1991; Tseng, McNeil, & Milenkovic, 1993; Murray, 1999; McNeil & Pratt, 2001; Hula & McNeil, 2008). The limited processing has also been

attributed to impaired working memory (WM) as the factor that causes or exacerbates the language-specific deficit (e.g. Caplan & Waters, 1999; Wright, Downey, Gravier, Love, & Shapiro, 2007; Ivanova, Dragoy, & Kuptsova, 2015).

While cognitive factors such as WM have been actively, frequently and relatively systematically examined in PWA, processing speed as a time-related factor, has not been examined systematically as a potentially critical factor affecting their language impairments. In addition, processing speed has not been investigated based on the information processing factors that could inform the locus and nature of the slowing.

Studies on slowed speed of information processing have been actively investigated in aging research, and a processing speed factor has been considered as a robust predictor of age-related cognitive decline (Salthouse & Ferrer-Caja, 2003; Eckert, 2011). Two accounts of information processing speed have proposed the features and mechanisms of slowness; providing different perspectives on slow processing speed associated with normal aging (Cerella, 1980; 1985, Salthouse, 1991; 1996). Cerella proposed a general slowing mechanism whereby the elderly are generally slower than young adults, and this slowing crosses task domains (e.g. in terms of lexical and non-lexical items). Consistent with this account, slower reaction times in the elderly was predicted by the young adults' reaction times as a function of task difficulty with the aging adults demonstrating slowness across tasks at a consistent ratio to the young adults. This was interpreted as supporting continuity across populations that is consistent with a "natural" aging process.

Salthouse (1991; 1996) hypothesized that aging is related to the reduction in speed of perceptual-motor processing and that this degraded speed performance causes the observed decreased cognitive functioning. He proposed "*limited time*" and "*simultaneity*" mechanisms in order to account for the nature of slowed processing speed as a function of aging. According to the

*limited time* mechanism, when reduced or limited time is allowed for processing certain information (i.e., compressed time presentation or concurrent conditions<sup>1</sup>), the time to perform later operations will not be sufficient due to the occupied portion of earlier operations. The *simultaneity* mechanism assumes that slow speed leads to a reduction in the amount of simultaneously available or active information. The key idea of this mechanism is that simultaneous availability of information diminishes due to decay or displacement (Salthouse, 1996; 2005). In this account, early information that has been processed is lost when later information is processed.

The general slowing account (Cerella, 1980; 1985) and speed of processing account (Salthouse, 1991; 1996) represent different aspects of the speed of information processing. General slowing is measured by the total amount of time spent completing each task (generally measured by reaction times) as a dependent variable, while processing speed account compares the number of correct items completed within certain time limits. The general slowing account provides time information, while the processing speed account provides the amount of information correctly processed. These two different approaches provide relevant methods for the detection and measurement of slowing and their impact on information processing.

With few exceptions, the age-related slowing studies in the information processing literature are based on low-level perceptual-motor processing speed performance. While there are a few studies of slowness in PWA that have investigated the physiological (e.g., slowed evoked potentials; Spironelli, Angrilli, & Pertile (2008)), and the perceptual-motor levels of information

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<sup>1</sup> The impact of a limited time mechanism can be measured in a dual-task condition because overlapped or conflicted information processing between early and later operations will reflect the slowed processing (c.f., Hula & McNeil, 2008).

processing (e.g., increased gap-detection times; Divenyi & Robinson, (1989); Stefanatos, Braitman, & Madigan (2007)), the research has not focused on the perceptual-motor processing level that requires limited cognitive resources and difficult computations. In addition, the research has not focused on potentially different sources of slowing in PWA. That is, the previous studies have specifically focused on the deficits encountered with slowed processing rather than the characterization of general slowing or the sources of slowing.

Assuming that PWA are slow, if their slowness occurs at the perceptual-motor level of information processing, the existing accounts of slowing (i.e., in terms of linguistic components or cognitive resources) should be reframed because the fundamental level of impaired information processing could potentially be the underlying source of slow performance on the language or other cognitive tasks. In other words, even though aphasia has been defined as a language disorder or a disorder of language related cognitive computations, it is still unclear whether the slow performance can be accounted for at a very basic perceptual-motor level that precedes more complex information processing and, further, whether the slowing is limited to the linguistic domain.

The primary goal of this study was to explore whether slow processing speed is aphasia specific. Based on the two different perspectives of general slowing and reduced processing speed, the slowness in information processing in PWA was explored using RT data.

The following specific issues were investigated: 1) whether the slowness is aphasia specific, based on the comparisons between people with left hemisphere damage who do not have aphasia (LHD) and people with left hemisphere damage who do have aphasia (PWA), 2) whether the slowness is specific to the nonlinguistic or linguistic domains; compared between normal healthy control (NHC) and PWA. The following questions were investigated in the current study:



1. Are there significant differences in the average reaction time per item among the average of the three assumed to be nonlinguistic tasks and the average of the three assumed to be linguistic tasks<sup>2</sup> among the three participant groups (PWA, LHD, and NHC)?
2. Are there significant differences in the magnitude of slowing between PWA and NHC, between PWA and LHD, and between LHD and NHC for each task?

RT data were plotted using Brinley plots<sup>3</sup>, which have been proposed as a method for measuring general slowing (this will be elaborated in the next chapter) to compare the magnitude of slowing in PWA compared to the other two groups.

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<sup>2</sup> The first nonlinguistic variable (Nonlinguistic task group 1) is derived from the average of the three reaction time tasks: Tapping, Simple perceptual-motor and Simple perceptual-motor + coordinated movement. The second nonlinguistic variable (Nonlinguistic task group 2) is derived from the average time per item for the correct pattern comparisons averaged across tasks 1 (easy: 30 items) and 2 (difficult: 30 items). The third nonlinguistic task (Nonlinguistic task group 3) is derived from the time per item for each of the 36 items on the RCPM. The first linguistic variable (linguistic task group 1) is derived from the average time per item for the correct letter comparisons averaged across tasks 1 (easy: 30 items) and 2 (difficult: 30 items). The second linguistic variable (linguistic task group 2) is derived from the average time per item for the correct lexical decision averaged across tasks 1 (easy: 30 items) and 2 (difficult: 30 items). The third linguistic task (linguistic task group 3) is derived from the time per item for each of the correct sentence completion tasks:

Nonlinguistic task group 1 – Tapping, Simple perceptual-motor function, Simple perceptual-motor and coordinated movement  
 Nonlinguistic task group 2 - Pattern comparison 1 and 2 (PA1 and PA2)  
 Nonlinguistic task group 3 – Raven’s Coloured Progressive Matrices (RCPM)  
 Linguistic task group 1 - Letter comparison 1 and 2 (LE1 and LE2)  
 Linguistic task group 2 - Lexical decision 1 and 2 (LX1 and LX2)  
 Linguistic task group 3 - Sentence completion (SC)

These task combinations (3 linguistic and 3 nonlinguistic task groups from a total of 11 tasks) were initially tentative. Exploratory factor analyses (principal component analysis (PCA)) were computed in order to identify the nature and interrelationships among tasks.

<sup>3</sup>Brinley plots typically involve plotting the mean response latencies of elderly adults against the mean latencies of younger adults (Perfect, 1994) on the same tasks and have been commonly used in aging research.

## **2.0 BACKGROUND AND SIGNIFICANCE**

### **2.1 BACKGROUND**

Slow processing speed is considered as one of the distinctive characteristics of PWA and evidence showing slow processing speed in language processing (e.g., slow response times, reaction times and reading times) has some support (Neto & Santos, 2012; Crerar & Alinson, 2004). An understanding of language performance and processing speed in PWA cannot be easily separated because language processing must be accomplished within a limited time frame in order to be successful. In this chapter, the slowing-related evidence will be reviewed for both linguistic and nonlinguistic performance in PWA. Furthermore, two information processing speed accounts, general slowing (Cerella et al, 1980) and speed of processing (Salthouse, 1996), will be reviewed regarding the purposes of the current study. .

#### **2.1.1 Slowness of Nonlinguistic Performance in PWA**

Numerous researchers have provided evidence of slowness for nonlinguistic processing performance in PWA. Efron (1963) proposed that temporal processing defects in discriminating sequential information might be the reason for the defects in language that requires sequential processes. Visual and auditory modalities were assessed. The visual stimuli consisted of 5 milliseconds of light (red and green-colored flashes), and the participants were instructed to decide which flash color appeared first (the intervals of time ranged from 0 to 600 milliseconds). The auditory stimuli consisted of high and low pitched tones presented for 10 milliseconds. The

participants were instructed to indicate which tone was first. The PWA were significantly slower on both auditory and visual sequence discrimination than the controls. Individuals with “expressive” aphasia performed significantly slower on the auditory modality for sequence discrimination, while those with “receptive” aphasia performed significantly slower on the visual modality for sequence discrimination. The findings indicated that those PWA had deficits (slowness) in processing nonlinguistic information, such as the discrimination of temporal sequences in both the visual and auditory modalities. However, Efron’s (1963) study does not provide clear information about where the temporal sequence deficits arise. That is, it has to be more clearly investigated whether the meaning of temporal processing deficits is at the level of slowed neuronal transmission, slowed signal detection, slowed response selection, slowed movement of the response, or overall slowed processing speed.

Chedru, Bastard & Efron (1978) specifically tested nonlinguistic tasks, pitch and micropattern (which is composed of two pure tones) discrimination tasks in people with fluent and nonfluent aphasia, and people with right hemispheric lesions who were not aphasic, as well as normal controls. The people with non-fluent aphasia demonstrated normal performance for both pure tones and micropatterns, however, individuals with fluent aphasia revealed a significant deficit (% correct) only in the micropattern discrimination test. The patients with right temporal lesions also demonstrated significant deficits in the micropattern test. The results indicate that PWA exhibited both linguistic and nonlinguistic processing impairments. However, since the nonfluent participants were not different from the normal controls, these nonlinguistic deficits do not appear to account for the aphasic deficits per se. Likewise, the fact that the right hemisphere damaged nonaphasic participants also demonstrated deficits on these nonlinguistic tasks challenges the aphasia specificity of these results.

Divenyi & Robinson (1989) also examined nonlinguistic auditory capabilities with various psychophysical tests in order to compare the patterns of auditory performance in 11 left CVA PWA, 4 right CVA participants without aphasia, and 8 age-matched healthy controls.

The results demonstrated that nonlinguistic auditory patterns could predict linguistic auditory comprehension in left CVA PWA. Auditory comprehension was strongly correlated to some of the nonlinguistic tasks: frequency discrimination ( $r = -.64$ ) and frequency uncertainty ( $r = -.80$ ). More importantly, there was a significant difference in performance between left CVA PWA and age-matched controls for most nonlinguistic auditory tasks except for the detection of tones in noise with and without frequency uncertainty.

This study is consistent with the notion that PWA demonstrate nonlinguistic processing deficits as well as linguistic deficits, and the presence of nonlinguistic dysfunction is consistent with the findings from Efron (1963). Furthermore, Divenyi & Robinson (1989) found a significant and high correlation between the nonlinguistic and linguistic performance in PWA. Thus, they proposed that this finding supports the contention that it may be important to consider the presence of nonlinguistic deficits in measuring linguistic abilities in PWA.

Laures (2005) investigated reaction time and accuracy for linguistic and nonlinguistic tasks in PWA and age-matched controls while performing an auditory vigilance task. In the linguistic task, low frequency words were presented auditorily, and the participants pressed a button to a predetermined target word. In the nonlinguistic task, four different pure tones were presented, one of which was the target sound to which the participants responded. These tasks required a relatively

low level of cognitive processing demands and represented processing speed based on a four-choice reaction time paradigm<sup>4</sup>.

The results revealed that reaction times for the PWA were not significantly different from the controls; however, accuracy was significantly lower in the PWA group. Importantly, there was no significant accuracy difference between linguistic and nonlinguistic domains for either group. For the PWA, correlations between severity, measured by the WAB AQ score, and reaction time ( $r = -.47$  nonlinguistic,  $r = -.66$  linguistic) were negative and moderate, and between severity and accuracy ( $r = .75$  nonlinguistic,  $r = .32$  linguistic) were positive and moderate to high. The pattern of the results reflecting no difference in reaction time between PWA and normal controls may represent a speed-accuracy trade-off for the normal controls, although this possibility was not discussed in the study. In addition, Laures (2005) claimed that the study's finding extends an hypothesized inefficiency of attentional processing in the linguistic domain (McNeil et al, 1991) to the nonlinguistic domain as well. However, this study (Laures, 2005) did not evaluate the complexity within the nonlinguistic and linguistic domains. That is, the vigilance tasks required a relatively simple level of information processing and a more complex level of tasks was not included in the study.

Another recent study (Villard & Kiran, 2014) investigated evidence for nonlinguistic slowing in PWA. One of their aims, which directly relates to the current study, was to examine the complexity effect on reaction times and between-session intra-individual variability (BS-IIV)<sup>5</sup> for

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<sup>4</sup> In the four-choice reaction time paradigm, each trial had a 2\*2 grid and each box was presented sequentially on a computer screen.

<sup>5</sup> In order to measure BS-IIV, five coefficients of variation (COVs) were computed for each subject, and each COV used the following formula:  $COV = \frac{s(\text{Session } i \text{ mean raw RT})}{x(\text{Session } i \text{ mean raw RT})}$ ,  $i = \text{Session } 1-4$ , Mean raw RT = mean raw RT for correct "E"/"R" responses between 350 ms and 2500 ms.

non-linguistic attention stimuli in PWA and age-matched controls. The nonlinguistic domain was tested using five different conditions<sup>6</sup>: 1) sustained visual attention, 2) sustained auditory attention, 3) selective visual attention, 4) selective auditory attention, and 5) auditory/visual integrational attention.

They hypothesized that there would be a task complexity effect on reaction times for both PWA and controls. However, they found that PWA exhibited the task complexity effect on both reaction times and BS-IIV, while age-matched controls showed the complexity effect on reaction times but not on the BS-IIV measure. Specifically, PWA exhibited two patterns: 1) Their reaction times were significantly slower on selective attention than on sustained attention, and 2) their reaction times were slower on auditory attention than on visual attention even though the slowing was not evident for every single PWA. This finding supported the claim that PWA demonstrate nonlinguistic domain slowing, and their attentional system deficits crossed visual and auditory modalities. While their primary goal of the study was not to compare between two groups, they found a significant main effect for group, but there was no significant group by condition interaction effect.

Importantly, Villard & Kiran (2014) investigated only the nonlinguistic domain and did not provide information on the relationship of these deficits with linguistic domain deficits. Thus, how this nonlinguistic slowing might be related to linguistic slowing was not investigated in this

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<sup>6</sup> Sustained Visual Attention: only visual stimuli were present, and visual instruction

Sustained Auditory Attention: only auditory stimuli were present.

Selective Visual Attention: visual and auditory stimuli were presented simultaneously.

Selective Auditory Attention: visual and auditory stimuli were presented simultaneously.

Integrational Auditory and Visual Attention: visual and auditory stimuli were presented simultaneously.

study. Considering that nonlinguistic processing may also be comorbidly impaired with linguistic deficits in PWA, an important issue would be whether the impaired functions cross-linguistic and nonlinguistic domains as well as auditory and visual modalities.

These studies' findings on nonlinguistic deficits indicate several critical issues about information processing in PWA. First, at least as measured by group effects, some PWA demonstrates nonlinguistic as well as linguistic-information processing deficits. Second, the relationship of those nonlinguistic deficits to the disruption of linguistic processing in PWA remains an open question. Third, PWA are significantly more affected by the rate (e.g. ISIs and stimulus durations) at which information is presented than normal controls (Hula & McNeil, 2008).

While slowness is a frequently observed attribute of PWA, studies have not investigated whether these linguistic or nonlinguistic deficits are specific to aphasia or whether they are the result of brain damage more generally or left hemisphere brain damage more specifically. Therefore, a more detailed investigation is necessary to examine those issues regarding the observed slowness in PWA.

### **2.1.2 Slowness of Language Performance in PWA**

Studies investigating slowness of language performance in PWA have been examined from several different perspectives. One aspect of this research has explored which part of the linguistic system (e.g., syntactic computation, lexical or phonological activation) is slowed. Slowness has also been approached from a limited or inefficient cognitive resources perspective as well as from a linguistic slowing account. Several studies have examined how PWA are affected by time-expanded/compressed speech as well as by the manipulation of overall presentation rates. More

studies have investigated the impact of time expansion, than time compression. The most relevant studies are reviewed below.

### **2.1.2.1 Loci within the Functional Architecture-Linguistic-Based Accounts**

The delayed lexical access (DLA) hypothesis (Love et al., 2008) and slow syntax hypothesis (Haarmann & Kolk, 1991; Piñango, 1999, 2000; Burkhardt, Pinango, & Wang, 2003) are two accounts addressing the slow processing of specific linguistic components in PWA<sup>7</sup>. Both accounts focus on the specific linguistic component that is slowed and neither examines the specific mechanisms of the slowing beyond speculating that neural activation is slowed for the unique linguistic computation, but without speculation or evidence of why it would be selectively impaired. Weak syntax (Avrutin, 2006) is another linguistic-based account that is similar to the other two. According to this account, damage in Broca's area decreases the amount of resources for powering syntactic operations. However the claim does not explain the nature of the reduced resources or how they translate into the mechanisms of slowness, only its location in the anatomical and functional architecture.

Love et al. (2008) criticized the slow syntax hypothesis in that it missed the possibility that the slowness could occur even before the syntactic level of processing is engaged. That is, it might be possible that lexical activation cannot be completed within a normal time frame before considering the possibility of the slowness at the syntactic stage. This account could also be overlapped with the resource account's claim that allocating or distributing attentional or working memory (WM) resources should occur within the given timeframe. In addition, the logic of the

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<sup>7</sup> Other accounts of slow language processing in PWA (i.e., limited or inefficient resource) will be reviewed after linguistic-based accounts.



Love et al argument about the slow lexical activation can be applied to information processing at other stages or levels. In other words, it may be possible that slowness could occur before the lexical level or during its interactive processing at semantic, morphologic, phonological, perceptual or motor levels of information processing.

The linguistic hypotheses use the speed or time concept to interpret the results or evidence of slow lexical or syntactic operations even though the concept overlaps with resource accounts (i.e., such as working memory or attentional resources). For example, in the weak syntax account, Avrutin (2006) proposed an economy mechanism to account for impaired syntactic operations in people with Broca's aphasia. He suggested that an economy hierarchy differs between impaired and unimpaired users. That is, PWA cannot use the most economical option in language processing because the damage to Broca's area makes the language operation more resource demanding, while people with unimpaired language have options in syntactic operations. This resource account implicates the involvement of cognitive resources in syntactic computation when PWA demonstrate weakened or impaired syntax.

Among these linguistic accounts, the DLA hypothesis provides a relatively clear explanation in comparison to the others. More specifically, according to the DLA hypothesis, slowed lexical access feeds slow syntactic processing, which causes a breakdown in automatic syntactic computation in PWA. When non-canonical (gap-filling) sentences were presented at a normal speech rate (in the context of a Cross Modal Lexical Priming (CMLP) paradigm), slowed lexical activation, as evidenced by reaction time performance, was observed in PWA (Love et al, 2008; Experiment 1; Ferrill et al, 2010; Experiment 1). However, PWA showed a normal pattern of reactivation when the same sentences were presented at a slowed speech rate (Love et al, 2008; Experiment 2; Ferrill et al, 2010; Experiment 2). Specifically, Love et al. (2008) conducted three

experiments in order to test their DLA hypothesis in people with Broca's aphasia. Experiment 1 investigated whether PWA demonstrate slow performance in lexical activation and reactivation at a normal speech rate (4.47 syllables per second). PWA demonstrated slowed lexical activation and reactivation for the auditory sentence presentations, while NHC did not show the slowed pattern under the normal rate. In experiment 2, a slower rate of auditory sentences (3.4 syllables per second) was presented for the same task as experiment 1. The PWA exhibited a normal reactivation pattern under the slower presentation rate. That is, this study found that PWA showed slow lexical activation and reactivation at the 4-6 syllables per second presentation rate, but they showed a normal reactivation pattern at the slower presentation rate<sup>8</sup>.

The third experiment focused on the performance of PWA in an off-line measure at both the normal and slowed speed of presentation. The people with Broca's aphasia showed improved performance at slower rates of speech. Their accuracy rate improved from 61% to 71% in the non-canonical sentence structures (passive/object-relative). The controls performed marginally, though significantly worse on these structures at the slower rate (97%) than at the normal rate (99%).

These hypotheses (DLA, slowed or weak syntax) have at least two issues or possibilities to be considered. 1) Neither of these hypotheses addresses the specific mechanisms of the slowness, but only tries to specify "where" in the cognitive architecture it is located. 2) The linguistic accounts do not address the possibility of slowness caused by reduced or inefficient available attentional resources. DLA cannot explain the evidence itself without a resource or other

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<sup>8</sup> PWA showed delayed lexical activations in experiment 2, while the reactivation pattern was not delayed. NHC showed disrupted processing of syntactic dependencies under the slow rate of auditory presentation.

account. Love et al (2008) also mentioned the possibility of the involvement of reduced resources<sup>9</sup> in order to explain the benefits of slowed speech input in the off-line sentence comprehension task (Experiment 3). They claimed that limited processing resources are involved in the disruption of the mechanism for building syntactic dependencies in real time, and these limitations affect the performance under the slowed speech input. These linguistic accounts (either slowed lexical or slowed or weak syntax) cannot fully explain how slowing speech can improve processing or comprehension without appealing to a mechanism that affects processing speed and ultimately to a source for the reduced speed itself. The relationship between slowing and attentional resources has been discussed (Hula & McNeil, 2008).

In addition, these accounts have neglected the possibility of general slowness of information processing in PWA at a fundamental level that is not process or linguistic domain specific (i.e., lexical or syntax). The possibility of general slowing has been mentioned in several studies (Friederici & Kilborn, 1989; Haarmann & Kolk, 1991) to support their argument; however, these authors did not provide clear evidence or directly investigate a source or mechanism for the general slowness of processing. It is possible that there is a general slowing at the perceptual-motor level of processing in PWA even before the phonological, lexical or syntactic level of slowing, which might affect higher (semantic, phonological, lexical or syntactic) levels of linguistic computation.

According to a review of the extant literature (McNeil, 1988), there are numerous deficits resulting from lateralized hemispheric brain damage that may accompany aphasia. The list

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<sup>9</sup> More importantly, the resource does not have to be reduced in quantity, even though they specifically mentioned it as reduced resource. It could be poorly allocated or distributed (McNeil, Odell, & Tseng, 1991).

includes sensory, motor, and low-level cognitive functions (i.e., increased simple and choice reaction times) as well as psychosocial and basic neurophysiological processes (i.e. increased performance time and general reduced efficiency). Therefore, if PWA were demonstrated to have a slowing of performance for perceptual-motor level processing tasks that could be demonstrate to be causally related to their linguistic deficits, a reconstructed linguistic account would be required regarding the origin of the language slowing.

#### **2.1.2.2 Neuro-Cognitive Mechanisms-Resource-Based Accounts**

##### **2.1.2.2.1 Electrophysiological time studies in PWA**

Numerous neurophysiological studies (McNeil, 1983a, 1983b; Kitade et al., 1999; Swaab et al., 1997; Dobel et al., 2002; Becker & Reinvang, 2007; Kielar, Meltzer-Asscher, & Thompson, 2012) have shown that PWA perform significantly more slowly and less accurately than age-matched controls on nonlinguistic tasks (McNeil, 1983a, 1983b) in addition to the expected poorer performance on linguistic tasks (Kitade et al., 1999; Swaab et al., 1997; Dobel et al., 2002; Rockstroh et al., 2004; Becker & Reinvang, 2007; Kielar, Meltzer-Asscher, & Thompson, 2012). In addition, PWA have been demonstrated to be slow at relatively low-level linguistic processing tasks involving syllable detection (Becker & Reinvang, 2007), word-category judgments (syntactic and semantic violation) (Wassenaar & Hagoort, 2005), as well as at high level processing tasks involving sentence comprehension using grammaticality judgments (Kielar, Meltzer-Asscher, & Thompson, 2012).

##### **2.1.2.2.2 Linguistic & nonlinguistic electrophysiological evidence of slowing in PWA**

Several electrophysiological studies have provided evidence that PWA are physiologically slow. Kitade et al (1999) reported that N400 latency in PWA was significantly longer than the control

group on a lexical decision task (meaningful/meaningless words in Kana, Japanese characters) under a visual oddball paradigm. Swaab et al (1997) also found a significantly delayed and reduced amplitude in the N400 evoked response for spoken sentence presented at a normal speed in PWA compared to age-matched controls. Half of the sentences had a semantically matched final word, while the other half had a semantically unmatched final word. PWA with moderate to severe deficits in comprehension showed a significant reduction in amplitude and delay in speed in the N400 potential. High comprehenders exhibited a similar N400 effect pattern to the age-matched unimpaired subjects. Becker and Reinvang (2007) found that PWA revealed reduced N1 and N2 amplitude for the syllable /ba:/. They concluded that the changed patterns in the early time window (N1 and N2) in PWA might be important for them to process complex language.

Wassenaar & Hagoort (2005) reported a delayed P600/Syntactic positive shift (SPS) effect<sup>10</sup> on word-category violations in PWA using an event-related potential experimental paradigm (ERP). They tested left hemisphere lesioned Broca aphasic patients, non-aphasic patients with a right hemisphere (RH) lesion, and age-matched controls on the sensitivity to the violation of word-category. They found that the two non-aphasic control groups were sensitive to the violation showing a clear P600/SPS effect, while the Broca aphasics exhibited a significantly reduced amplitude and delayed P600/SPS effect. Importantly, this study attempted to control for the “cerebral lesion” effect by including a right hemisphere lesioned control group. However, there may be a reason to postulate that the left hemisphere is specialized for or more sensitive to temporal constraints on information processing than the right hemisphere and hence their control group may

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<sup>10</sup> P600/SPS here indicates two syntax-related ERP effects: an anterior negativity (referred to as LAN: Left Anterior Negativity) and posterior positivity.

not have served as the best test of “brain damage” as an alternative explanation for impaired linguistic functions.

Electrophysiological evidence of slowing has also been shown in the nonlinguistic domain in PWA. Greenberg & Metting (1974) investigated the averaged encephalic responses (AER) in PWA between linguistic (words) and nonlinguistic stimuli (white noise). Participants’ communication abilities were evaluated based on the Functional Communication Profile. The evoked potentials were also recorded at right and left hemisphere placements. Significant differences in latencies were found between hemispheres, but only for those participants who had severe communication difficulties. That is, PWA with severe communication difficulties showed shorter AER latencies from the right than from the left hemisphere. However, PWA with mild to moderate communication difficulties did not show significant differences in AER latencies between hemispheres. Interestingly, there were no significant differences between linguistic and nonlinguistic stimuli. However, they claimed that the results on linguistic vs. nonlinguistic stimuli are inconsistent because their previous study (Ratliff & Greenberg, 1972) found that nonlinguistic stimuli had longer latencies than the linguistic stimuli. Therefore, the comparison between linguistic and nonlinguistic processing requires replication.

McNeil et al (1983a; 1983b) also investigated an auditory processing factor (intensity) for both linguistic and nonlinguistic stimuli in PWA and normal controls. The purpose of the 1983a study was to evaluate the effects of increased intensity to both ears (diotic) in PWA. Three supra-threshold intensity levels were tested: 70, 85, and 100 dB SPL. The dependent variables were auditory-evoked response (AER) amplitudes and latencies for the N2 and P1 responses. The stimuli were short bursts of speech noise. Correlate variables included a nonverbal (short bursts of

speech noise) intensity-sequencing test (NVIST), a word-sequencing test (Minimally Varied Phoneme Test; MVP), and the Revised Token Test (RTT).

Interhemispheric AER differences revealed the mean latency from the left hemisphere for the N2 response was longer than that from the right hemisphere, but AER latencies and amplitudes in the damaged hemisphere were not significantly different from those in the intact hemisphere at all intensities in PWA. There was also no significant difference in the performance of NVIST, MVT, and RTT at any intensity level. Although the overall results were not significant, this pattern is different from other previous studies (Kolman & Shimizu, 1970; Ruhm, 1971; McCandless, 1978) showing that AER from the damaged hemisphere was significantly longer in latency as well as reduced in amplitude compared to the intact hemisphere.

The McNeil et al (1983b) tested a neurological extinction model using stimulus intensity. According to the model, information travels faster to the intact than the impaired hemisphere, and this causes extinction and disruption in message delivery. To compensate for this between-ear timing difference, the intensity was increased by 15 or 30 dB above the other ear. They hypothesized that the selective amplification to the damaged hemisphere's contralateral ear would decrease the stimulus arrival time and also increase the amplitude relative to the intact hemisphere. The results showed that intensity was traded with time as expected. However, the patterns of the results were not consistent or clear. Most statistical results were not significant, even though the patterns of the results exhibited the reduction of the extinction to the impaired hemisphere. However, regardless of the statistical significance, overall patterns of the two studies by McNeil et al (1983a; 1983b) provide inconclusive evidence of physiological slowing for nonlinguistic versus linguistic processing in the auditory modality in PWA.

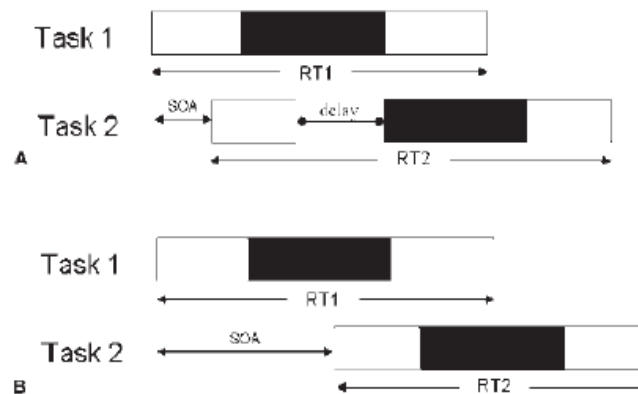
### **2.1.2.3 Psychological theories and evidence of slowing in PWA**

McNeil, Odell, and Tseng (1991) proposed a general resource theory to account for a range of aphasic behaviors. They hypothesized that a poorly allocated pool of attentional resources for linguistic computations causes incomplete processing at various stages of language representation building, which slows down the system. According to their hypothesis, either inadequately distributed or inefficient allocated attentional resources could cause incomplete or impaired language processing in PWA, and the allocation control system can be disrupted for several reasons. One reason might be intermittent attention allocation caused by fluctuating of biological rhythms or encephalographic patterns. Another reason might include an insufficient allocation of attention caused by an inappropriate estimation for the demand of tasks within the system. This attentional resource allocation account takes a view that a slowed system is the output of unequally distributed or inefficient attentional resources; however, this view of the disrupted attentional control system is not consistent with the notion of a fundamentally slowed processing system in PWA.

Hula and McNeil (2008) investigated whether the slowness of language processing in PWA is consistent with a central bottleneck (CB) model of attentional distribution. As shown in Figure 1, under dual-task conditions (which essentially represent all language computations) a serial processing bottleneck leads to a delay in the central processing of a second task. That is, when there is a stimulus-onset asynchrony (SOA) between two tasks that is short, Task 1 and Task 2 are more overlapped. More specifically, in a short SOA condition (Figure 1. A), the central processing of Task 2 exhibits delay, and the reaction time for Task 2 is increased relative to a longer SOA (Figure 1. B) because the central processing (i.e., the darkened area in Figure 1) part is overlapped between Task 1 and Task 2. This impact of different SOAs on reaction times (increased or slowed



reaction times for the short SOA condition relative to the long SOA condition) is called the Psychological Refractory Period (PRP) effect. In the long SOA condition (Figure 1. B), the central processing of the two tasks is not overlapped and causes no delay in the central processing of Task 2.



**Figure 1. Processing delay or slowness explained by central processing under Central Bottleneck (CB)**

#### **Model**

(A: SHORT SOA, B: LONG SOA)  
(Hula & McNeil, 2008)

This explanation can be interpreted to mean that PWA need longer SOAs, even in a sentence (not just in traditional dual tasks) to integrate each part. The authors argued that this resource and bottleneck explanation potentially accounts for “the moment-to-moment breakdown in language performance and timing deficits in information processing that can explain the generation of specific linguistic impairments” (p. 184). This explanation is consistent with the general idea of processing speed account (Salthouse, 1996), which suggests that the slow speed of processing mediates cognitive performance because the slower speed causes the reduction in “the amount of simultaneously available information” (Salthouse, 1994, p. 258). The difference here is

that Salthouse suggests that the neurophysiological consequence of aging causes the slowness, whereas Hula and McNeil suggest that deficits in the central bottleneck, resulting from the focal lesion within the language network causes the processing problems. These authors also argued that the PRP account was not inconsistent with the resource allocation account.

This account (Hula & McNeil, 2008) may be important because it shows that PWA exhibit different performance from normal controls depending on a time or speed factor (i.e., SOAs). Although the evidence and this line of reasoning propose a processing speed mechanism for the deficits in PWA, it is based on evidence generated from one specific paradigm (i.e., the dual-task paradigm). Accordingly, additional research is required to verify the generalizability of the finding, as well as the specificity of effects in terms of populations, domains of knowledge affected, and experimental tasks that reveal such slowing effects.

Campbell and McNeil (1985) examined if and why a slow rate of speech presentation causes better auditory comprehension in children with acquired language disorders. They pointed out that a number of researchers have found that the information processing system in PWA is slow, but their conclusion provides limited information about the nature of the slowed information processing. Most studies on time manipulation in the nineteen seventies and nineteen eighties did not offer a clear theoretical framework to explain the effects of the stimulus presentation rate on information processing (mostly auditory comprehension) in PWA. Campbell and McNeil (1985), however, used Kahneman's (1973) model of attention and information processing capacity to study auditory comprehension in children with acquired language disorders. According to their claim, time-expanded signals allow individuals to allocate attention in a more efficient way and lead to better auditory comprehension performance. The framework proposed by the original model (Kahneman, 1973) assumes that there is a pool of attention that is limited but not fixed. In

the theory, the central pool shares attentional resources across all modalities and cognitive processes (e.g., sensory, motor, and integrative). Due to limitations of the attentional resources, competition in processing exists when two or more stimuli or cognitive operations occur simultaneously. When spare attentional resources are available because they are not needed for a primary task, they can be made available for a secondary task or several additional tasks. Campbell and McNeil (1985) found that their results were consistent with the limited attentional capacity model. They observed that the mean scores on a secondary task in a dual-task auditory sentence comprehension paradigm improved, for both language-disordered and normal groups, when the primary task was time-expanded compared to when it was not time-expanded<sup>11</sup>. Consistent with Kahneman's (1973) limited attentional capacity model, the results of this study attributed the improved auditory comprehension in the sentences presented at a normal rate concurrently with the sentences in the expanded time condition, to the availability of additional attentional resources as a function of the additional time available for their deployment.

There are also studies based upon temporal and spectral processing or central auditory processing resources (Eberwein et al., 2007). The effects of both time compression and expansion on auditory language processing have been investigated. According to this notion, a slower rate of presentation facilitates the auditory comprehension in PWA because it compensates for a slowed system that is not available to them at a normal rate of speech. However, this account, like the

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<sup>11</sup> For the primary task sentences, sentences of the Revised Token Test (RTT) were used (e.g. "Touch the blue circle and green square" and "Touch the big blue circle and little green square"). For the secondary tasks, sentences with the same level of vocabulary, syntactic complexity, and length as the primary stimuli were used (e.g., "Touch the grey button and brown box" and "Touch the little grey button and the big brown box"). In the first condition, both tasks were presented at a normal rate. In the second condition, the primary task was expanded 75% in time, and the second task was presented at a normal rate.

linguistic account above, is based on the assumption that PWA are slow, but what is slowed<sup>12</sup> is undefined other than an impaired mechanism for distributing attentional resources.

According to the study by Eberwein et al (2007), time-compressed sentences tax the central auditory processing system, while a time-expansion allows more efficiently allocated attentional resources. This account is consistent with the hypothesis offered by McNeil, Odell, and Tseng (1991).

There are, however, other potential explanations for the effects found in the work of Campbell and McNeil (and others). That is, it is unclear whether attentional resources is the only component of information processing that is related to processing efficiency, or whether other components can explain the results. For example, the improved performance in the second task may not be because of spare attentional capacity from the primary task. It might be the case that the expanded time for the primary task was the best fit or timing required for their auditory comprehension. That is, if their processing speed mechanism is slowed, the expanded time is necessary to process the information from the primary task. The expanded time would provide the best timing<sup>13</sup> or speed that allows them to process all of the required information from the primary task. In this case, there would be no delayed processing within the primary task, and the secondary task can be performed normally because all the processing for the primary task has already occurred, and the time available is adequate for processing the secondary task. Thus, the results

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<sup>12</sup> Love et al (2008) and other studies that were reviewed in the linguistic account (Chapter 2.1.1.1) pointed to the locus of the slowing that was not addressed in these studies. However, these studies do not offer a satisfactory mechanism for the slowing.

<sup>13</sup> Popove & Poova (2015) described this best timing with a different terminology, which is “spatio-temporal opportunity windows” specifically related to neuronal activity coordinated by brain oscillations. The neuronal oscillations provide a modulatory function of integrating information.

could be interpreted as a timing disorder or a processing speed mechanism that is closely related to attentional resource allocation.

If the processing system is generally slow in PWA, it is necessary to consider first what is involved in the information processing system or its components. In most information processing studies (specifically on normal healthy controls including the aging population), the efficiency of information processing is examined with tasks that require both perceptual and motor functions. The possibility that slowing could occur at earlier and at more fundamental levels of information processing has not been adequately investigated, even though attentional resource allocation and working memory could be limited and those limitations could negatively impact language performance in PWA.

Both the resource and linguistic accounts for aphasia require, or might be augmented by the integration of speed of processing (SOP) factors. Information processing speed is measured by using time limits imposed on the stimulus (i.e., inter-stimulus-interval (ISI), stimulus-onset asynchrony (SOA), compression rates, and presentation time) or time limits imposed on the response (i.e., reaction times or the time span limiting the number of correct responses per unit of time).

Kolk and van Grunsven (1985) proposed a time-based account for agrammatic deficits. Several other similar studies (Haarmann & Kolk, 1994; Kolk, 1995; Haarmann, Just, & Carpenter, 1997) followed. In this view, agrammatic comprehension in PWA is an issue of disrupted timing. It is assumed that building syntactic representations requires some amount of time to reach a required level of activation and that activation must be maintained long enough to allow integration across linguistic units. They proposed that either slow activation or fast decay will cause slowed syntactic processing. The “slow activation” case yields a longer time to arrive at the required

activation threshold. The “fast decay” case happens when the processing of the elements reaches the critical threshold level but is not maintained long enough to allow integration with other information in the sentence.

These two accounts could be a hybrid claim between the linguistic account and the resource account. In addition, these are overlapped with underlying mechanisms from the processing speed account (Salthouse, 1996) as well as other psychological theories of memory such as the decay theory or the interference theory. In addition, this time disorder account has been proposed to explain specifically language processing. It ignores the possibility of a general slowing at simple or fundamental cognitive processing levels in PWA.

#### **2.1.2.4 Time-manipulated Studies: Time Expansion and Time Compression in PWA**

The majority of time-manipulated studies (with the possible exception of the Campbell and McNeil 1985 study), including both time expansion and compression studies in PWA were not motivated by an explicitly stated theoretical views of information processing. Thus, these studies have evaluated whether the time-manipulation improved or degraded performance and have speculated about the locus of the effects within the linguistic information processing system. However, the origin or nature of slowness has been investigated rarely. The time-manipulated speech (most often time expanded speech) has demonstrated inconsistent effects across studies. Some studies have found improved language comprehension with expanded durations (Suci, 1969; Fillenbaum, 1971; Salvatore, 1974, 1978; Liles & Brookshire, 1975; Lasky et al., 1976; Hageman & Lewis, 1983; Nicholas & Brookshire, 1986; Pashek & Brookshire, 1982), while others reported that the benefit of time expansion was limited (Blumstein et al., 1985; Blanchard & Prescott, 1980); often using the same dependent measures.

Lasky et al. (1976) examined the effects of rates of auditory presentation and pause intervals on the comprehension of sentences with varying levels of syntactic complexity. Fifteen PWA received active-affirmative (e.g., The mother is splashing the baby.), active-negative (e.g., “The mother is not splashing the baby.”), and passive-affirmative (e.g., “The baby was splashed by the mother.”) sentences at rates of 150 words per minute (wpm) with a 1-sec inter-phrase pause time (IPT) at constituent boundaries; 150 wpm with no pause; 120 wpm with a 1-sec IPT; and 120 wpm with no pause. Participants demonstrated significantly better comprehension (% correct) at the slow presentation rate than at the normal rate and the greatest comprehension when the slower rate of presentation and IPT intervals were combined. The percentage of correct items was the highest at the slower condition (120 wpm) with pause and the lowest under the 150 wpm conditions without pause in the active and passive sentence types. Performance was similar for the negative sentence type. PWA exhibited better understanding in active and passive sentences than in negative sentences and they were better at the slower rate than at the normal rate. The author concluded that the presentation at the slower rate with pause was the most beneficial condition for auditory comprehension in PWA. However, Lasky et al. (1976) did not clearly explain why PWA exhibited better performance with IPT at the slower rate of presentation. They suggested that the pause intervals take a role as cues for linguistic and perceptual dissection, and also suggested that the pauses provide for extra processing time at the syntactic level. However, it is still unclear what mechanisms are involved in the benefits provided by the slower presentations. This explanation might assume that PWA have slowed processing speed and need additional time for better performance. In order to clarify this connection between slowing mechanisms and the benefits from the additional time, understanding and examining a processing speed factor in PWA is required.

Similar to Lasky et al. (1976), Blumstein et al. (1985) tested the effects of slow speech on auditory comprehension for PWA. They investigated the effects of slow speech at three stages of language processing by increasing the vowel segments of each word (vowel level), inserting silences between words (word level), and inserting silences within phrases at constituent boundaries (syntactic level). More specifically, the sentences (180 wpm) were constructed based on the following conditions. First, an examiner recorded the sentences naturally at a slow speaking rate (180 wpm to 110 wpm). Second, word durations were increased in the sentence by adding 70-100 msec to the vowels (180 wpm to 110 wpm). Third, word segmentation extended the sentences by adding 250-msec silent intervals between the words (180 wpm to 110 wpm). Fourth, silent intervals were inserted at salient syntactic boundaries (180 wpm to 110 wpm). In simple sentences, 1,000 msec was added between the noun phrase (NP) and verb phrase (VP). In more complex sentences, the silence was inserted at the embedded clause. Also, 500 msec was added between the verb (V) and noun phrase (NP) of/within the verb phrase (VP) (e.g., The boy chased [silent interval] the girl). The duration inserted was different depending on the sentence complexity. Thirty-four PWA (Broca (5), Conduction (5), Wernicke (6), Global (9), and other (9) types) participated in this study, as well as four control subjects. The task was to choose one of three pictures that matched with the sentence they heard. The findings showed that PWA showed the significantly improved performance at the slower rate, but only at the syntactic level. Moreover, only the Wernicke's group improved at the syntactic level of processing at the slower rate. These results indicate that additional time facilitated language comprehension in some individuals with aphasia, although the effect was limited to the syntax level of language and only for the Wernicke aphasia type. The authors analyzed the effects across types based on a high and low comprehension ability to determine if slow speech is related to the severity of comprehension impairments. There



was no significant difference between the high and low comprehension groups in the effects of slowed speech. The authors concluded that the results of this study generally supported the concept that slow speech facilitates auditory language comprehension for PWA, even if the effect was limited to the sentence level and to the Wernicke's type. However, they did not provide direct evidence for slowed processing in PWA or that the benefits of the slowed rate were either limited to the language domain or that it was aphasia specific. They concluded that additional research is needed in order to explain the "time-facilitation effect" or the "no time-facilitation effect" in PWA.

Several studies have also used IPT to assess the facilitation of auditory comprehension in PWA (Suci, 1969; Fillenbaum, 1971; Salvatore, 1974, 1978; Liles & Brookshire, 1975; Hageman & Lewis, 1983). These studies found that additional time is helpful to improve the performance of auditory comprehension in PWA. However, like those of Lasky et al and Blumstein et al, those studies do not provide clear evidence for a slowed language processing in PWA.

Other time-manipulated studies used compression and expansion for a whole sentence instead of inserting additional pause time between the linguistic components or by lengthening specific phonemic segments. Bergman et al. (1977) used both expanded and compressed speech to examine the role of stimulus speed on auditory comprehension. PWA received an unspecified version of a token test while listening to three different rates of presentation (e.g., Fast: 50% compressed, Moderate: 25% compressed, Slow: 35% expanded). An electric rotary-head speech compressor-expander was utilized for the speed alteration. The slow speed (with 35% expanded rates) of speech presentation did not lead to significantly improved performance in PWA, while the performance was lowest under the fast speed condition. That is, the mean accuracy was not significantly different between the moderate (time compression) and slow rates (time expansion), while there was a significant difference in accuracy between fast and moderately fast rates (50%

and 25% compression) in the PWA. The PWA were sensitive (performed worse) to fast rates, but they did not benefit from slow rates. Normal controls did not show significant difference across conditions.

Blanchard and Prescott (1980) studied the effect of temporal expansion of the Revised Token Test stimuli (as recorded by McNeil, 1977) in healthy controls and PWA. They investigated four different rate conditions-150 wpm (originally recorded rate: 2.5 words per second), 113 wpm (temporally expanded by a factor 1.25), 75 wpm (temporally expanded by a factor 1.5) and 37 wpm (temporally expanded by a factor 1.75) expanded levels. The authors hypothesized that temporal expansion would have beneficial effects on auditory processing in PWA. Contrary to their prediction, the expanded speech conditions were only moderately different from the control condition in both accuracy and latency for both groups. They reported that the differences in the actual score between expansion conditions were minimal; however, there was a statistically significant improvement in accuracy for some of the participants. RTT means for the normal healthy control group was 14.64 (no expansion), 14.65 (at expansion factor 1.25), 14.62 (at expansion factor 1.50), and 14.64 (at expansion factor 1.75). The RTT means for PWA group was 12.59 (no expansion), 12.76 (at expansion factor 1.25), 12.91 (at expansion factor 1.50), and 12.84 (at expansion factor 1.75). Therefore, the expanded condition did not substantially affect the performance in either PWA or normal controls. There was a significant interaction between the groups and the expansions, so normal controls performed statistically significantly more accurately than the PWA. However, accuracy and latencies were not significantly different across rate conditions for either the PWA or the normal healthy controls.

Pashek and Brookshire (1982) investigated the accuracy of sentence comprehension under slowed (120 wpm) and normal rates of speech (150 wpm) with/without linguistic stress at the

critical parts in the sentences (Token Test derived from DeRenzi & Faglioni, 1978 stimuli). The four formats of stimuli presented to the PWA and controls were: 1) slow rate with exaggerated stress, 2) slow rate with normal stress, 3) normal rate with exaggerated stress, and 4) normal rate with normal stress. There was no significant interaction between rate and stress. Accuracy was significantly higher at the slow compared to the normal speech rate in PWA. PWA were divided into two subgroups: high and low auditory comprehension groups, based on significantly different overall accuracy scores on the DeRenzi & Faglioni (1978) version of the Token Test. The accuracy was significantly higher with exaggerated stress than without the stress in PWA, but only for the PWA with high performance. Accuracy was not different across speech rates or stress conditions in the normal controls. This is probably because a lack of test sensitivity for normal controls. For the positive effects of slowed rate in PWA, the authors proposed a possibility that PWA process more slowly on auditory stimuli than controls. According to their account, a mismatch may occur between the rate of stimulus presentation and PWA's processing rate, when normal rates are presented. Thus, auditory comprehension could be facilitated when the rate of presentation is slowed because the rate might approximate the PWA's optimal processing rate. This possibility assumes that PWA have slowed processing that affects their performance. However, this study like the many others did not address the fundamental concept or mechanisms of slowed processing in PWA and their results provide only tertiary evidence for slowing.

As reviewed in this chapter, there have been several studies on language processing and speed of stimulus presentation in PWA. Whether the time-manipulated speech (especially time-expanded speech) had a predictable impact on language performance was inconsistent across the studies. Some studies found improved language comprehension with additional time (Suci, 1969; Fillenbaum, 1970; Salvatore, 1974, 1978; Liles & Brookshire, 1975; Lasky et al., 1976; Hageman

& Lewis, 1983; Nicholas & Brookshire, 1986; Pashek & Brookshire, 1982), but others reported that the benefit of time-expansion was limited (Blumstein et al., 1985; Blanchard & Prescott, 1980). While these time- or speed-related studies were predicated on slowed processing in PWA, they do not provide evidence for a slowed linguistic processor as an explanation for the language impairment. Indeed, normal controls also benefited from presentation rates in some studies and they are not assumed to have slow linguistic processing.

In order to explain the phenomena caused by expansion or compression, an understanding of the underlying mechanisms of the slowing in PWA is critical. Additionally, a closer link between the observed phenomenon and the psychological, or neurophysiological mechanisms are worthy of exploration. The following two psychological constructs from the information-processing literature offer one account.

## **2.2 INFORMATION PROCESSING HYPOTHESES**

### **2.2.1 General Slowing**

The general slowing claim originated from a mathematical theory of communication or information processing proposed by Shannon (1948) and Shannon & Weaver (1949). This communication theory has been applied to information processing theory in psychology by Miller & Frick (1949); Miller (1956); Miller, Galanter & Pribram (1960), and many others.

Two human performance principles formalized as, Hick-Hyman's Law (Hick, 1952; Hyman, 1953) and Fitts Law (Fitts, 1954; Fitts & Peterson, 1964), are based on this classic

information theory. Both laws explain the relationship between response time and task complexity (Sleimen-Malkoun, Temprado & Berton, 2013). Card et al (1985) characterized these two laws as representing basic perceptual (Hick-Hyman's Law) and motor (Fitts Law) principles. Hick-Hyman's law has been used to account for the choice-reaction time (CRT) task results and Fitts law has been used to explain sensory-motor (or peripheral components) performance such as rapid aiming-movements (cited by Seow, 2005 and Sleimen-Malkoun et al., 2013).

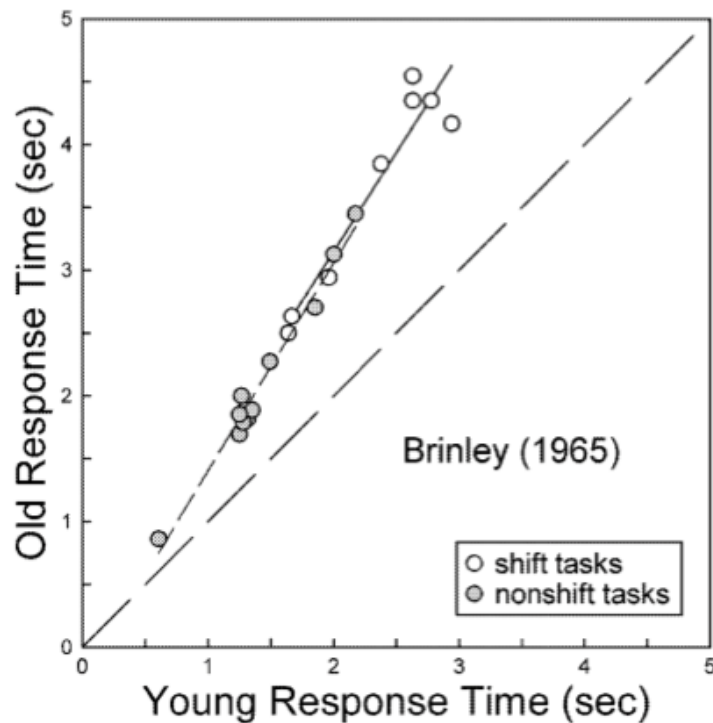
According to Hick-Hyman's Law, response time (RT) is linearly related to the "index of difficulty" (ID):  $RT = a + b * ID$  (a and b are constants), and its slope reflects the efficiency function (EF) of the CNS's information processing. Thus, the slope of the EF is a measure of central processing.

Information processing speed can be tested by such simple movement tasks as moving from a resting position to a target position (Sleimen-Malkoun et al., 2013). According to Fitts law, movement time (MT) is linearly related to the width and distance of the target (ID), which is measured as follows:  $ID = \log_2(2 \times D/W)$  (W: The width (*W*), D: distance of the target). The linear relationship between MT and ID is referred as Fitts law.

These two basic principles of human behaviors derived from classic information theory have been applied to such fields as human-computer interaction and cognitive psychology and such problems as general slowing as a function of aging.

The general slowing account (Cerella et al., 1980; 1985; Hale et al., 1987) has been proposed to explain commonly observed age-related decline in the speed of information processing. The strength and influence of the claim has been influenced greatly by the results of the meta-analyses that combined the data (reaction time data) from several studies by using a variety of information processing tasks, such as Sternberg memory scanning, stimulus-response

mapping, multiple choice reaction time, alerting reaction time, card-sorting, line length discrimination, paired associate learning, and stimulus-response recall (Cerella et al., 1980). In this general slowing account, elderly adults demonstrate a proportional slowing across mental functions; simple sensorimotor, as well as higher cognitive processing. Cerella et al (1980) collected the data from 18 studies that used 99 different information processing tasks for their meta-analysis. The data were analyzed using a Brinley function (or Brinley plots), which has been commonly used in meta-analyses of cognitive aging (Cerella, Poon, & Williams, 1980; Cerella, 1985, 1990; Cerella & Hale, 1994). In the Brinley function, the mean response times are calculated between two groups (e.g., young and elderly) for each separate condition in an experiment. One group's mean response times for each condition are plotted on the corresponding other group's mean response times (Figure 2). The main claim from this study is that there is general age-related slowing in information processing.



**Figure 2. Plotted mean response time of older and younger groups in the corresponding condition  
(reconstructed by Myerson et al., 2003)**

Brinley's (1965) study was not originally designed to examine the general slowing account, rather the study was aimed at determining whether the two different age groups (young and old) were differentially affected by the presence of task shifting. Cerella et al (1980) applied the Brinley plots in order to address the general slowing. According to the study results, the latencies of elderly adults were predicted by the young adults' latencies (See Figure 3) (Cerella et al., 1980).

When the reaction times of the elderly adults were plotted together with the reaction times of the young adults as shown in Figure 3, from Cerella et al. (1980), a simple proportionality between the elderly and young adults was found. This approximately linear relationship was found between young and elderly adults' response times by using a simple linear regression formula as follows.

$$[(E1) Y = 1.36X - 0.7]$$

The correlation was .95 (accounting for about 90% of variance) with the elderly data being predicted by the young adults' data. They also tested whether there were task effects based on knowledge of task type. They found that the contribution of task type was not significant. When the age group was divided into over 60 and between 30 and 60, the percentage of age-specific variance accounted for increased from 40% to 78% as well as the increase in the percentage of overall accountable variance (90.2% to 96.4%). Regression equations were different for the two age groups as follows.

a. Over 60 [(E2)  $Y = 1.62X - .13$ ]

b. Under 60 (but over 30) [(E3)  $Y = 1.16X - .04$ ]

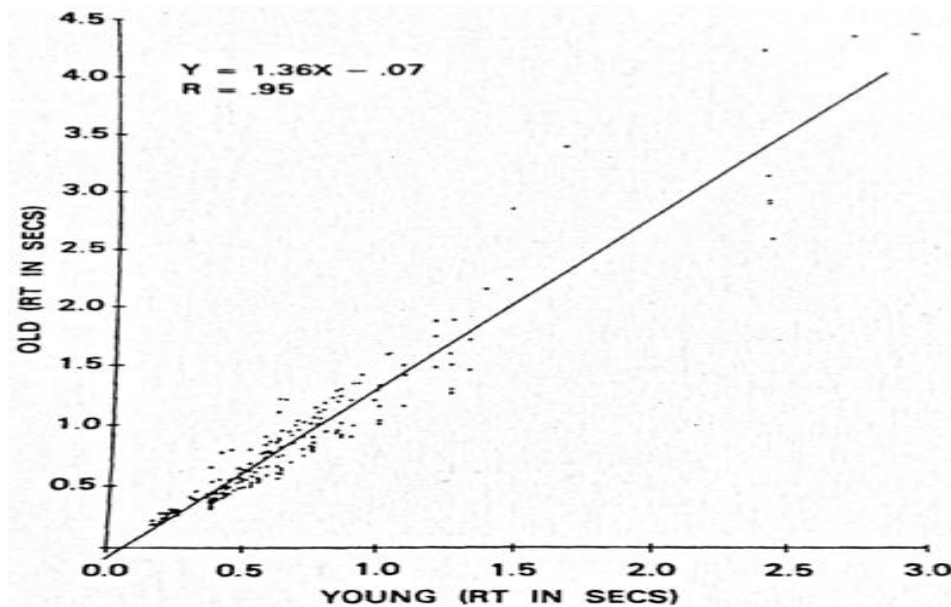


Figure 3. Reaction times comparison between young and elderly groups (from Cerella et al, 1980)



The general slowing hypothesis has been tested in several subsequent studies (Cerella, 1985; Hale et al., 1987; Madden, 1989; Lima et al., 1991) to examine whether slowing is found across cognitive tasks, and populations. The patterns of the general slowing results from Cerella et al (1980) were replicated by Hale et al (1987). They found consistent results with the general slowing account across tasks, but the general slowing pattern was accelerated in the healthy elderly compared to the young healthy adults. Second, general slowing has been also investigated in Specific Language Impairment (SLI) (Kail, 1994; Windsor & Hwang, 1999; Windsor et al., 2001) and children with normal development (Hale, 1990). Several studies found general cognitive slowing for different components of lexical and non-lexical tasks for SLI (Lima et al., 1991; Kail, 1994; Hale, 1990; Hale et al., 1991). Other studies have reported evidence of specific slowing depending on the analysis methods for SLI children (Windsor et al., 2001; Windsor & Hwang, 1999). Windsor et al (2001) found that general slowing in SLI has been identified, and they reported that there was general slowing across models or methods (i.e., a proportional, linear, and nonlinear model), but the slowing did not appear for all SLI individuals. That is, even though most SLIs performed slowly, some did not show slower performance than the chronological age-matched (CA) group.

Based on the premise that a comparison between lexical and non-lexical domains might provide important insight into the general slowing mechanisms, studies have compared these domains in normal aging adults. Lima and Myerson (1991) found that the slope was steeper in the non-lexical domain than in the lexical domain even though general slowing appeared for both domains. This indicated that processing the non-lexical information was slower than the lexical domain in the older population (age: 65-75) that was tested. Hale et al (1987) found a linear slope between the middle to elderly (age: 50-75) and young adults (age: 20-25) for nonverbal tasks such

as choice reaction time, letter classification, mental rotation, and abstract matching. All the tasks were plotted on the same slope with a linear slope. The elderly adults (age: 65-75) were 1.25 times slower than the young adults, and middle age adults (age: 50-60) were 1.12 times slower than the young adults. Therefore, there was an aging effect on information processing speed. Madden (1989) also reported that there was a linear slope between the young and elderly groups for word/nonword discrimination tasks. Two experiments were conducted with the same stimuli in the study, and those were with two different words per second (wps) (Experiment 1: 2 wps, Experiment 2: 5wps). The slopes were 1.53 and 1.43 for each experiment between elderly and young adults. So Madden (1989) concluded that the presentation rate (wps) didn't significantly affect the results of two experiments.

Thus, general slowing has been consistently found in several different populations across different tasks as an effect of normal aging. However, the source of slowing has not been addressed. That is, there could be slowing due to aging (maturation) and slowing due to a language disorder in the SLI population.

### **2.2.1.1 Measurement and Components of General Slowing**

General slowing has been measured by a variety of reaction time paradigms, with early studies using choice reaction times to measure components of information processing. Reaction times are frequently analyzed with Brinley plots in order to calculate efficiency functions between groups.

Cerella (1985), for example, used choice reaction time tasks requiring different types of cognitive operations such as classification of playing cards, memory scanning, stimulus decoding, stimulus discrimination, letter classification, mental rotation, abstract matching, stimulus-response mapping, card-sorting, line length discrimination, paired associate learning, and stimulus-response recall. General slowing has been demonstrated across a large range of cognitive operations.

## 2.2.2 Processing Speed Account

Following Salthouse's (1991; 1996) proposal of a general reduction in processing speed to account for differences (aging effect) between young and old healthy adults, many researchers across different disciplines have investigated processing speed. The specific mechanisms underlying the processing speed account, however, remain debated. Thus, this review will discuss the mechanisms subtending the processing speed account, as well as neurological evidence for an age-related reduction of processing speed.

### 2.2.2.1 Neurological Background of Cognitive Processing Speed

Eckert (2011) conducted an extensive review of the age-related neurobiological slowdown underlying processing speed. According to his review, age-related slowing in processing speed is related to global cortical declines in neuropil (Morries & McManus, 1991), total gray matter volume (Chee et al., 2009), gray matter volume decline in the frontal cortex (Kochunov et al., 2010; Eckert et al., 2010), loss of myelination (Morries & McManus, 1991; Fjell & Walhovd, 2010), reduced volume of the prefrontal cortex (Kennedy & Raz, 2005), changes in cerebellar morphology (the midline surface area of vermis: declive, folium, tuber region) (MacLulich et al., 2004) and reduced total cerebellar gray matter volume (Paul et al., 2009). Importantly, based on this review, using an independent component analysis (ICA) using *source-based morphometry*<sup>14</sup>, neurobiological evidence for age-related changes in processing speed has been found in frontal

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<sup>14</sup> According to Eckert (2011), source-based morphometry compares groups by using a structural imaging analysis. Specifically, it correlates with a specific measure and the estimated volume of voxels with common covariance. He also pointed out that this approach is useful to identify unique effects and reduces the necessity of the number of comparisons.

and cerebellar gray matter. Source based morphometry analyses seek unique correlations of changes in frontal and cerebellar regions with processing speed tasks. Eckert identified several patterns in gray matter that correlated with processing speed. The changes in frontal and cerebellar gray matter regions were identified as unique predictors of processing speed.

Neurological research has provided a potential connection between processing speed and LHD. Specifically, damage in white matter in the left hemisphere has been linked to deficits of processing speed (Turken et al., 2008). According to this study, the structural integrity of white matter tracts was related to processing speed deficits. Turken and colleagues investigated the effect of white matter lesions on cognitive processing speed in the patients with left-hemisphere damage<sup>15</sup>. They found that simple cognitive processing speed<sup>16</sup> assessed by the Digit-Symbol test (subtest from WAIS- III) was associated with the structural integrity of white matter. The strongest relationship to the performance in processing speed was found in the left parietal white matter.

While there appears to be some connection between aphasia and processing speed, it is not clear whether this connection is due to a left hemisphere lesion more generally, or whether it is related more specifically to the language impairment (aphasia specific). That is, it is not clear whether the speed factor is related to left-hemisphere damage or to damage in the processing speed-related neural systems subtending language-specific cognitive operations. Given the literature reviewed, it appears more likely that the slowing would be related to the hemisphere damage and not the presence or absence of aphasia. However, the participants in the studies that have included brain-damaged participants have been inadequately selected (e.g. right hemisphere

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<sup>15</sup> Turken et al (2008) did not provide specific information whether their LHD participants had aphasia or not.

<sup>16</sup> Simple cognitive processing speed (e.g., the digit-symbol test) does not require high levels of cognitive processes that most language tasks require.

damaged control groups) and characterized to confidently interpret the findings relative to lesion versus language specificity.

#### **2.2.2.2 Processing Speed Hypothesis**

Since Birren (1974) first theorized slowness underlying cognitive decline with age, over several decades Salthouse has provided considerable evidence showing slower processing speed as a function of aging. According to the processing resource view, the speed of information processing is a significant source of age-related decline in cognition. Fast speed in cognitive operations (or fast rehearsal, in Kail & Salthouse, 1994) allows the processing of greater amounts of information per unit of time. In this view, processing speed is one type of a processing resource (Salthouse, 1991, p. 325).

According to the processing speed account as proposed by Salthouse (1996), reduced processing speed leads to dysfunction or impairments in cognition. More specifically, the main idea of the claim is that aging is related to decreased speed across many different processes and the reduced speed causes degraded cognitive functioning. Either relevant information processing cannot be completed successfully (*limited time*) or the information processed early is not available for later information processing (*simultaneity*).

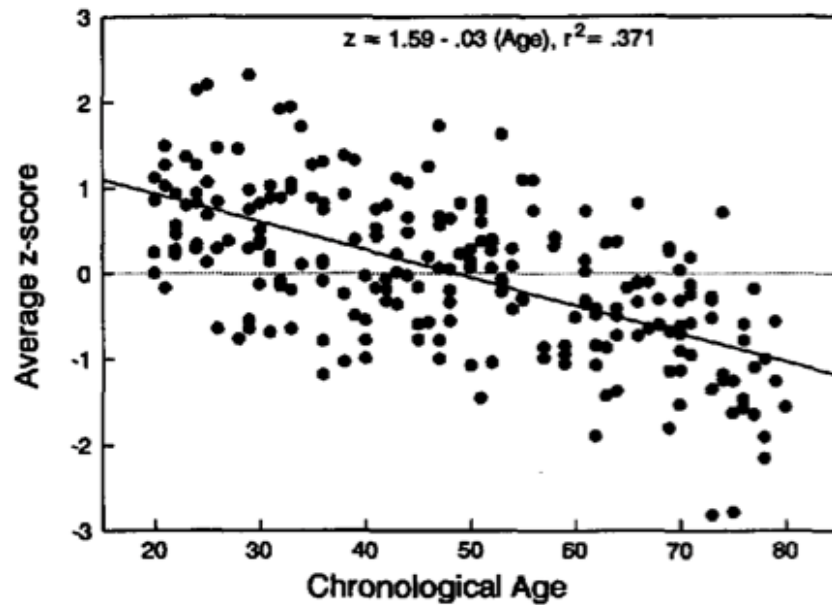


Figure 4. The relationship between age and composite scores of processing speed (from Salthouse, 1996, data is originally from Salthouse, 1993)

Figure 4 shows age-related slowing on two perceptual-motor tasks (i.e., Letter Comparison and Pattern Comparison tasks) with 221 participants from 20 to 80 years of age. The slope shows a significant decrease in speed of performance with increased age, though the percent variance accounted for ( $r^2 = .371$ ) is relatively limited.

### 2.2.2.3 Limited Time and Simultaneity Mechanisms

Two key mechanisms, the limited time mechanism and the simultaneity mechanism, have been used to account for the observed speed and reduced cognitive functioning (Salthouse, 1996). These two mechanisms are summarized in

Table 1 related to their underlying assumed mechanisms, how they are applied or explained and a brief statement about the source or evidence for them.

The simultaneity mechanism represents another attempt to explain the relationship between processing speed and cognitive performance. The key idea of this mechanism is that simultaneous availability of information diminishes due to decay or displacement (Salthouse, 1996; 2005). In this account, early information is lost when later information is processed. Accordingly, processing deficits are due to the gap between the time missing from the early information processed and the time it takes for the later information to finish processing. Those simultaneous processes overlap, and relevant information is no longer available when it is needed. In this view, inefficient processing could occur due to the dynamics of slow speed of information activation or fast rate of loss, decay or forgetting (see Salthouse, 2005, for a review).

As evidence of the simultaneity mechanism, Salthouse provided results showing that statistical control of speed measures resulted in a significant reduction in age-related variance in working memory performance (Salthouse, 1996). More specifically, an average of 77.6% of age-related variance was reduced after statistical control on reaction time measures and an average of 85.1% was reduced after the control on perceptual speed measures. Thus, processing speed functions as a mediator between age and cognition, and no pure motor speed was accounted for in this study.

**Table 1. Comparisons between limited time mechanism and simultaneity mechanism**

	Limited time mechanism	Simultaneity mechanism
Mechanisms & Principles	<p>1) Relevant cognitive operations are executed too slowly to be successfully completed in the available time.</p> <p>2) The time to perform later operations is greatly limited when a large proportion of the available time is occupied by the execution of early operations.</p> <p>3) More processing frequently results in better performance, and the opportunity to accomplish a large amount of processing is greater when the speed of processing is faster.</p>	<p>1) Slow processing reduces the amount of simultaneously available information needed for higher level processing.</p> <p>2) The simultaneous availability of relevant information: The product of early processing may be lost by the time that later processing is completed.</p> <p>3) Information (quantity or quality) decreases in availability over time as a function of either decay or displacement. Slower speed of activating or processing information rather than the rate of information loss or decay</p>
Applications	<p>If someone has fast or slow processing speed, information processing might be as follows.</p> <p>With fast processing speed (under the limited time mechanism),          ➔More amount of information (accuracy: number of correct items) can be processed within the time limit.          ➔More successful processing (high accuracy) is available within the time limit.</p> <p>With slow processing speed (under limited time frame),          ➔Less amount of information can be processed within the time limit.          ➔Less successful processing (incomplete processing) could occur within the time limit.</p>	
Evidence	<p>The effect of manipulation in the presentation duration or the rate of presentation, such as the experiment by Kersten and Salthouse (unpublished paper, 1993)</p>	<p>The concept of working memory is another way to measure this simultaneously activated information (Salthouse, 1994, for a review). Therefore, showing significant reduced age-related variance in working memory performance after the statistical control of speed measures could be the evidence of how the simultaneity mechanism works with the processing speed factor in aging population (Salthouse, 1996)</p>

The concept of working memory is another way to explain the simultaneity mechanism, which involves the amount of simultaneously active information (Salthouse, 1996). The simultaneity mechanism remains uncertain. That is, it is still unknown whether working memory tasks are the most appropriate for assessing the amount of simultaneously available information



and whether the reduction in the amount of simultaneously active information comes from the slow activation or a loss of information over time. The results of Love et al (2008), showing normal reactivation in PWA for some linguistic operations, seems to argue for slow activation rather than a loss of information over time. Regardless of the limitation, Salthouse noted that this simultaneity mechanism is still a plausible candidate as a psychological explanation for reduced cognitive functions with aging and as a source for slowing.

It might be confusing to differentiate the *limited time* mechanism from the concept of the *simultaneity* mechanism. The limited time mechanism is revealed through timed tests, while the simultaneity mechanism can operate and is measured without external time limits. Slower speed indicates that less processing can be completed within the given amount of time, and faster processing speed leads to higher levels of performance (the number of correct items within a given time limit). In order to accomplish high-level cognitive operations such as association and integration (operations necessary for most language tasks), all of the relevant information should be simultaneously (or nearly simultaneously) available. Thus, the time available to activate and maintain the information for the moment-to-moment processing is important relative to the simultaneity mechanism.

There are limitations to the speed of processing accounts. First, two speed of processing mechanisms were proposed to explain how the processing speed functions, but those were not enough to explain how the two mechanisms provide differential predictions. Second, the accounts for the mechanisms are not clearly differentiated from other cognitive factors, such as WM. This is probably because the concept of the processing speed overlaps with a vast portion of memory mechanisms (Salthouse & Coon, 1993; Salthouse, 1994; 1996), or the concept of processing speed

underlies other cognitive elements. Accordingly, it is necessary to clarify and perhaps reconstruct the concept of processing speed as proposed by Salthouse and others.

While these two processing speed accounts provide an experimental method for evaluating slow processing, they do not offer much insight into the mechanisms for the slowing either with aging or pathology. Accordingly, the two mechanisms are potentially relevant to the experimental paradigm, but they provide limited specification for the underlying cognitive or physiological mechanisms for the observed slowing.

#### **2.2.2.4 Measurement and Components of Processing Speed**

In the processing speed account, processing speed is measured with the amount of information (i.e., a number of correct items) completed within a given time limit. Thus, a typical method to assess processing speed is to use time limits (Eckert, 2011). A subject has to perform perceptual & cognitive tasks as quickly as possible and the number of correct items within the specified time limits represents the dependent variable with which to determine each subject's processing speed. In this paradigm, it is critical to determine what is slowed. An analysis of the tasks that have been previously studied offers a window into the slowed cognitive components. At the most simple motor-level, processing speed within limits can be assessed using a finger-tapping task whereby the number of taps produced within a given time period is measured.

Boxes, digit copying, letter comparison, and pattern comparison tests are frequently used processing speed measures. These tasks require simple cognitive operations. For example, for the boxes task, participants have to draw lines within a given amount of time in the gap between two lines in order to make a box figure. The number of correctly completed items within the prescribed time limits represents the processing speed performance. Digit copying requires copying digits as quickly as possible within the given timeframe. These two tasks involve heavily motor rather than

perceptual functions. Letter comparison and pattern comparison tasks are similar in format. In these tasks, participants determine whether two strings of letters or patterns are the same or different within a given time limit. These discrimination tasks are based more on perceptual than motor functions. These tasks have been reported to show good reliability for young and elderly populations (Earles et al., 1997; Salthouse & Babcock, 1991).

Within these experimental paradigms, basic levels of perceptual-motor functions are assessed to test processing speed. As is evident in the examples above, some of the tasks make greater demands on motor function, and others more on perceptual mechanisms. Other tasks can be utilized that require additional attention, memory, coordination, and other cognitive operations. A range of tasks is required in order to explore the locus and mechanisms of information processing and loci of slowed operations.

## **2.3 SUMMARY AND STATEMENT OF PURPOSE**

### **2.3.1 Summary**

Slowness in language performance has been considered as one of the main characteristics of PWA. They have shown slowness in overall language production, comprehension and on numerous cognitive performance tasks. As reviewed earlier, several studies have found that PWA exhibited slowing performance in the both nonlinguistic and linguistic domains. Other studies found that they were also slow in performance on electrophysiological measures as well as psychological measures. In addition, multiple studies with PWA have shown that their language performance is affected by time manipulation or time-related factors (presentation rate of the stimuli,

compressed/expanded stimuli, and ISI limitations). However, none of these studies has investigated if the slowing is specifically related to the presence of aphasia, as opposed to the presence of left-hemisphere brain damage or a general aging factor. Studies have also failed to assess a range of nonlinguistic and linguistic task difficulties to assess language specificity as a locus of the slowing in PWA.

Another critical point is that slowness on language tasks originating anywhere between sensory detection and motor execution could affect language performance. There is a possibility that slowed processing speed leads to the difficulties in language processing in PWA by breakdown or decreased efficiency of cognitive computations supporting language. The slowing could occur when complex linguistic computations are not engaged, such as at the simple perceptual-motor levels, or while engaged in more complex nonlinguistic tasks. If that is the case, then slowing per se does not seem to fully explain the linguistic-specific or linguistic-dominant impairments that define and characterize aphasia.

### **2.3.2 Statement of Purpose**

The current study aims to contribute to the understanding of the role that cognitive information processing speed plays in linguistic and nonlinguistic performance in PWA, LHD, and NHC. More specifically, this study is based on the conceptual and methodological backgrounds of information processing accounts, general slowing and processing speed; specifically for domain specificity and aphasia specificity. Some studies have found slowed processing of linguistic-specific information (slow lexical activation and slow syntax) but there has been little explanation for the mechanisms of the slowness in these accounts. Others have proposed mechanisms for the slowness such as attentional resource allocation deficits or bottlenecks during language processing; however, these

more mechanistic accounts have been less specific about the locus of the processing deficits. That is, it is unclear whether it is linguistic domain specific, and whether it is related to the left-hemisphere damage as well as whether the slowing involves motor, perceptual, or other cognitive functions. With few exceptions, the studies that have manipulated stimulus presentation time have focused on the effect of time expansion. That is, the goal of these studies was simply to determine whether PWA would benefit from slow presentation in their processing and ultimate comprehension; with the tacit assumption that if they did benefit, a slowed processing system could be inferred.

General slowing and processing speed accounts derived from the aging literature are reviewed in the previous chapter. According to this review, information processing speed in PWA can be investigated within the two general frameworks or methods.

Therefore, the current research is designed to contribute to an understanding of the presence and nature of slowing in PWA. The specific purposes of this study are to explore: 1) aphasia specificity, and 2) linguistic domain specificity.

Domain specificity was examined by assessing the performance between linguistic and nonlinguistic domains. More specifically, this question was to investigate whether PWA are significantly slower on linguistic processing tasks than on nonlinguistic tasks. The detailed information on the tasks and the methods of analysis are provided in section 3.0.

If processing speed is significantly slower in linguistic, compared to non-linguistic tasks in PWA, it would be interpreted as support for domain specific slowing (linguistically specific). However, whether slow processing of linguistic stimuli is uniquely impaired in PWA will be more specifically explored in the second purpose. If linguistic processing speed is significantly different

between PWA and LHD without aphasia, and if the LHD group performance is not different from the NHC group, it would imply that PWA's linguistic slowing is aphasia specific.

In order to examine aphasia specificity, both PWA having left-hemisphere damage (LHD) and people with LHD but without aphasia were compared on the same tasks. If processing speed is not aphasia specific, then their performance should not be different from the group with LHD without aphasia and both groups' performance should be significantly different from NHC. This result would be interpreted as evidence that reduced processing speed for language tasks is the result of the left-hemisphere damage and not associated with the aphasia per se. To assess whether any observed slowing is unique to having a LH lesion, the NHC and the two brain-impaired groups were compared. The effects of aging was not examined in this investigation, as age was controlled across groups

Finally, the different loci of slowing were compared using tasks that require various components of potential slowing sources. Each task represents different loci of slowing such as pure motor, perceptual-motor and perceptual-cognitive.

## **2.4 SIGNIFICANCE**

The current study is important for several reasons. First, it provides information on the potential sources of slowing (LH brain damage or aphasia) and loci (linguistic vs. nonlinguistic, versus domain general) of slowness in PWA by comparisons between the domains and among the three groups. Understanding the mechanism and locus of slowing in PWA contributes to the understanding of their language performance and possible mechanisms subtending it.

Second, this study provides information on what kinds or aspects of slowing contributes most to the performance in PWA using several kinds of tasks that represent different information processing domains and stages (Figure 5). As such, it investigates whether the slowing is derived from early processing, controlled processing, perceptual-motor functions or from a combination of these. Slowing at early processing stage of information processing is consistent with its contribution to slow language processing in PWA. Slowing on cognitively demanding and controlled processing is consistent with an impairment of controlled processing; a contributing factor to the aphasia.

Third, the results of this study have implications for aphasia treatment and for the measurement of treatment effects. As processing speed is an important underlying factor for language processing in PWA, it could be one target for aphasia treatment to improve language functions. Direct training of cognitive factors, such as working memory and attention, has shown limited effects on language functions to date using Attention Processing Training (Sohlberg, 1987; 2001, Coelho, 2005; Murray et al., 2006), and this may be because speed of processing underlies those other cognitive factors such as working memory and attention and has not been targeted or measured in these studies.

## **2.5 INFORMATION PROCESSING MODEL AND AN INFORMATION PROCESSING SCHEMATIC IN PWA**

There are two overarching views of language impairment in PWA: 1) those who attribute the linguistic impairment to focal dominant hemisphere damage that impairs the rules or

representations of the language, or 2) those who assign impaired cognitive functions (e.g., limited WM or poorly allocated or reduced attentional resources), usually attributed to damaged language-specific neural networks, with those cognitive dysfunctions causing the disruption in language processing. These two main views are based on the assumption that early processing stages, as well as input and signal (sensory) registration stages are not impaired or slowed and late processing stages such as response selection and output (sensorimotor control) stages are not impaired or slowed.

An information-processing schematic (see Figure 5) summarizes the specific tasks and some of the primary assumptions about their psychological and neurological underpinnings for this study. It is offered as a summary of methods and assumptions for the specific purposes of the current study and is not proposed as a general model of information processing for aphasia. This schematic is based on one of the classic human information processing models by Wickens (1992). In this classic approach, human information processing is compared to computer processing. Wickens (1992) model is a hybrid of the two approaches (the classic and ecological approaches), and his model emphasizes mental processes such as selecting, interpreting, retaining, or responding to the information input as well as feedback as human factors. However, this model does not explain linguistic & nonlinguistic components, and also the mental processes of the model are not specified in details sufficient for this investigation.

The conceptualization, reflected in Figure 5, is also inspired by the model of McNeil & Kimelman (1986) in terms of the concept that attention, prior knowledge, expectations and environment are involved at all levels of processing. However, in the current framework attention is served by information processing speed. In their model, McNeil and Kimelman, (1986), proposed that sensory reception and LTM (such as linguistic/nonlinguistic knowledge, situation



specific expectations (probabilities), and world knowledge) are intact in PWA. Psychophysiological processing and short-term auditory memory are primarily impaired, while attention and linguistic/nonlinguistic performance are impaired due to the primary impairments. Within the current framework, the components of early and controlled processing are more clearly specified and slowed early processing is assumed to lead to an additive impact on more demanding and controlled processing. It is also assumed that information processing speed affects every stage of information processing, from the early transmission of sensory information to motor execution. In other words, poor language performance in PWA could be due to slowed information detection, perception, activation, transmission, and integration as well as slowed motor functions.

Accordingly, if there is slowed processing in early stages, it should lead to subsequent, and perhaps additive breakdown in the later stages of information processing. Specifically, once the input signal is presented, the individual with aphasia registers the sensory (e.g., visual, auditory, or haptic) information. Their processing is assumed to be age-related normal up to the sensory registration and detection stages. Detection is defined as a process of identifying the presence of a stimulus (cited from [www.oxforddictionaries.com](http://www.oxforddictionaries.com)). In the early processing stage, simple cognitive operations (i.e., discrimination) are required, regardless of modality (visual or auditory) and regardless of the content of the information (linguistic/nonlinguistic). In this stage, STM for brief and few elements is required. Discrimination in this early processing stage can be defined as a process of clarifying a relation by comparison based on similarities and differences among stimuli ([www.oxforddictionaries.com](http://www.oxforddictionaries.com)).

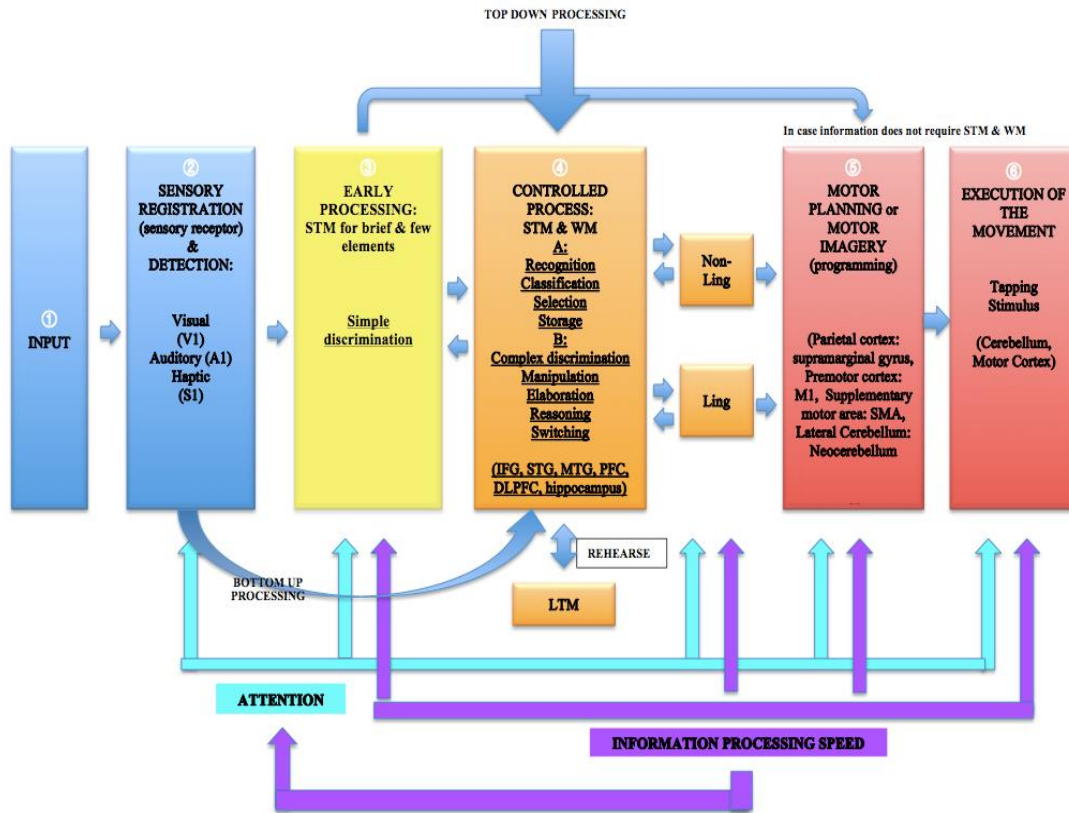


Figure 5. A schematic of information processing in PWA<sup>17</sup>

After this early processing stage, information is delivered to either a controlled processing stage or directly to the motor planning stage (as in simple reaction time tasks) depending on whether the information requires an additional higher level of processing or computation. If the information processing does not require heavy demands on recognition, classification, selection, storage, maintenance, manipulation, elaboration, reasoning, or switching, it moves to motor planning and execution stages. Recognition is defined as a process of identifying something, which

<sup>17</sup> Primary visual cortex (V1), primary auditory cortex (A1), primary somatosensory cortex (S1), inferior frontal gyrus (IFG), superior temporal gyrus (STG), middle temporal gyrus (MTG), prefrontal cortex (PFC), dorsolateral prefrontal cortex (DLPFC), Primary motor cortex (M1), and supplementary motor area (SMA).

is learned previously and is retrieved from memory ([www.oxforddictionaries.com](http://www.oxforddictionaries.com)). Classification indicates a process of associating something according to shared qualities, categories, or characteristics ([www.oxforddictionaries.com](http://www.oxforddictionaries.com)).

For this investigation, controlled processes are divided into two different kinds (A and B) in the schematic. The first type of controlled process requires STM. The early processing also uses STM, but the task demands are low: requiring only the comparison of two items. In controlled processing, the demands of STM are greater than that required in early processing where comparison of several items at the same time is required (Controlled processing A). Another kind of controlled process (Controlled processing B) requires WM. Some tasks (i.e., Raven, Sentence completion tasks) require more complex and controlled processing such as manipulation and switching of information than simply holding and recalling information.

In most previous views, PWA were assumed to have normal nonlinguistic processing, as well as normal early processing because aphasia has been defined as a language-specific disorder. However, for this study, aphasia is hypothesized to be an inefficient information processing disorder caused by slowing.

It is assumed that attention and processing speed may affect almost every stage of processing. Specifically, if information processing is slowed at an early processing stage, its effects will cascade through later stages of processing, which will also be slowed. In this instance, language processing will be affected by slowing at lower levels of processing. It was predicted that the impact of a delay or slowed processing at the controlling process stages (i.e., STM and WM) will have more consequential effects than at early processing stages.

Regarding attention, less efficiently or poorly distributed attentional resources will negatively affect each component of the information processing system. The processing speed

factor plays a role in supporting the overall attention factor. However, it cannot be determined whether or not slowed processing speed causes attentional deficits, without conducting experiments specifically designed for that purpose. It was beyond the scope of this investigation to explore this important relationship.

The current study was intended to serve as a first step to assess the possibility that PWA have impaired or slow processing for early low-level linguistic and nonlinguistic cognitive processes. In addition, slowing of information processing at different levels of complexity (simple motor to cognitively more complex linguistic and nonlinguistic tasks) was assessed.

### **2.5.1 Hypotheses**

**Hypothesis 1: Aphasia specificity:** PWA will demonstrate significantly slower and less accurate processing (longer RT and fewer number of correct items) than LHD and NHC on all processing speed tasks. Comparisons among the three groups will determine whether any observed slowness in PWA is aphasia-specific or whether it is attributable to a more general brain-damage-related slowing. General slowing with aging has been well-documented (Cerella, 1980; 1985). Thus, with the three groups being age-matched, the aging-related general cognitive slowing will be assumed to have been controlled. Any existing differences between the groups would indicate either aphasia-specific deficits or brain-damage-related slowing.

Case A: If there is an aphasia-specific deficit slowing, the NHC and LHD groups will not perform significantly differently from each other.

Case B: If all three groups perform significantly differently from each other ( $PWA > LHD$ ,  $PWA > NHC$ , and  $LHD > NHC$ ) then an aphasia-specific plus brain-damage-related deficit will be inferred.

Case C: If the slow performance in PWA is caused by left-hemisphere brain damage, then both PWA and LHD performance would demonstrate significantly slower performance than the NHC but the two left hemisphere damaged groups will not differ significantly from each other. This case would indicate that there is a brain-damage-related slowing and no aphasia-specific slowing.

These three cases (Case A-C) of aphasia specificity could be confounded depending on the domains. That is, PWA could be significantly slower than NHC and LHD on linguistic tasks, however, PWA might not be significantly different from LHD in nonlinguistic tasks. Aphasia specificity might exist only for the linguistic domain.

**Hypothesis 2: Domain specificity:** PWA will demonstrate linguistic specific deficits on timing measures (slower RT-ab and lower accuracy).

Aphasia is defined as a language-specific (domain-specific) disorder. If this definition is accurate, then PWA should exhibit significantly slower RT-ab and lower accuracy performance on the linguistic than the nonlinguistic tasks and those differences should be greater than any such difference for the other groups (LHD and NHC).

### 3.0 RESEARCH DESIGN AND METHODS

#### 3.1 PARTICIPANTS

Three participant groups were enrolled in this study: 1) thirty Normal Healthy Control (NHC), 2) fifteen Left Hemisphere Damaged (LHD) without aphasia and 3) fifteen LHD people with aphasia (PWA) (see Appendix E-G for the descriptive information of all the participants). The estimation of the number of required subjects for this study was calculated using the sample size software, PASS 13 with a multilevel model. Statistical power was set at .80, and an alpha level was set at .05 in the calculation. The provided effect size (between group effect size  $d=1.07$ , within group effect size  $d=0.361$ ) for the sample size calculation was based on the data from one previous study (Arvedson & McNeil, 1985). The previous study was chosen because 1) the task (lexical decision) was similar, and 2) both between and within groups were compared in the study. In order to verify the sample size from PASS, G-Power analyses were also calculated based on the same conditions (power and effect size), and the total sample size was estimated to be 15 participants per group (Total 60 participants). Therefore, data from a total of 60 participants (PWA: 15, LHD: 15, and NHC: 30) were collected in this study based on the crossed check on the sample size.

All participants met the following screening criteria: (a) native American English speakers, (b) at least 8 years of education, determined by self-reported questionnaire. Participants were also assessed for (c) normal or corrected vision with the reduced Snellen chart with 20/40 or better binocular visual acuity, (d) the immediate/delayed language recall task from the *Assessment Battery of Communication in Dementia* (ABCD) (Bayles & Tomoeda, 1993) with a ratio (the delayed recall/immediate recall  $\times 100$ ) greater than 0.70.

NHC reported a negative history for neurologic, limb motor, psychiatric, visual, speech/language, or reading impairments by self-report (APPENDIX D). Participants not meeting the screening criteria did not receive the experimental tasks.

The Rey-Osterrieth Complex Figure Task (ROCFT) (Rey, 1941) was provided as a nonlinguistic descriptive test for all participants.

### **3.2 LANGUAGE AND COGNITIVE SCREENING AND DESCRIPTIVE MEASURES**

The PWA group was tested for the presence<sup>18</sup> of aphasia using their performance on the Comprehensive Aphasia Test (CAT) (Swinburn, Porter, & Howard, 2004). Additionally, all participants were provided with the following language and cognitive descriptive measures: a) one form of the story retelling procedure (SRP) (McNeil et al., 2008) as an index of connected spoken language, b) forward digits-pointing span as a short-term memory measure, and c) sentence (cleft-subject and cleft-object) reading span, alphabet span, and subtract-2 (Waters & Caplan, 2003) as working memory measures.

All participants in the PWA and LHD groups also demonstrated evidence from medical records of a left hemisphere infarct. The evidence was derived from behavioral neurology and/or neuroradiological scans that have been interpreted as showing a unilateral left hemisphere lesion.

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<sup>18</sup> PWA were included in the study if they show a deficit in at least 1 subtest or summary score from at least 2 different language modalities (listening, speaking, reading, and writing) on the test. The presence of deficits was determined by pre-established cut-off scores for each subtest based on the definition of aphasia (McNeil & Pratt, 2001).

The NHC participants demonstrated performance that is within the established range of normal for the behavioral tests on which the pathological participants were included in the study.

### **3.3 EXPERIMENTAL DESIGN AND STIMULI**

In both general slowing and processing speed experiments, the tasks are usually similar, but the dependent variables for each are different. The experimental tasks require relatively simple cognitive operations, and are typically based on the perceptual-motor level of processing. Those tasks, however, did not completely address the research questions for the current study. In the current study, therefore, tasks with greater cognitive complexity or greater motor demands were also included as well as the simple perceptual-motor tasks.

The typical dependent variable with which to index general slowing is reaction time. The dependent variable typically used to quantify processing speed is the number of correct items within a given time limit. However, in order to use a common metric across all tasks, the time per stimulus (or item) was used in this study as the dependent variable instead of either RT-ab to complete each task or the number of correct items responded to within a given time limit. This variable was calculated based on both the RT-ab and the number of correct items ( $\text{time stimulus} = \text{RT-ab (total time spent in completing correct items for each task)} / \text{the number of correct items}$ ). The reason for using this combined variable is that each task provides RT and accuracy based on two different task procedures. Some tasks are presented with limited time (with accuracy as the dependent variable), and others do not have a time limit (time to complete each item provides the dependent variable). For example, the tapping task provides the number of taps in 10 seconds (time interval between the taps was chosen as the time variable from the CRTT-RT 1 task). This fixed



duration (10 seconds) cannot be compared to the time variable of other tasks without an imposed time limit. Thus, choosing a common variable across all the tasks is essential.

A total of 11 experimental tasks were included for this study (as shown in Table 3, Table 4 and Table 5): CRTT-RT 1: tapping; CRTT-RT 2: perceptual-motor RT; CRTT-RT 3: perceptual-motor with movement RT); Raven's Colored Progressive Matrices; Pattern comparison 1 and 2; Letter comparison 1 and 2; Lexical decision task 1 and 2; and Sentence completion task. These tasks were chosen to represent potentially different sources of slowing or different performance processes that might be differentially or selectively slowed.

Each of these tasks requires relatively simple motor or perceptual-motor processing, and some require greater demands on linguistic and nonlinguistic processing. However, each of the tasks represents potentially different sources of slowing. For example, CRTT 1-RT1 (tapping task) requires primarily or purely motor behavior. CRTT 2-RT2 requires simple perceptual-motor functions and CRTT 3-RT3 requires simple perceptual and RT processing plus coordinated movement. The Raven's Colored Progressive Matrices task (RCPM) has more perceptual than motor demands. In addition, RCPM, and Sentence completion tasks require more complex and controlled processing (component or stage 4 from Figure 5) than the pattern-comparison, letter comparison, and lexical decision tasks (Table 4 and Table 5).

The tasks can also be classified as linguistic or non-linguistic for the research questions of the current study. According to this classification, the letter comparison, the word/non-word lexical decision tasks, and sentence completion task represent linguistic processing speed tasks. The RCPM, the three simple reaction/response time tasks (CRTT RT1-3), and the pattern comparison task were chosen to represent non-linguistic processing tasks.

The experimental letter comparison and pattern comparison tasks were derived from the perceptual-motor processing speed tasks previously used by Salthouse (1991; 1996) (obtained directly from Salthouse: personal communication). These tasks are based on simple cognitive operations such as pattern or letter comparisons, and do not require complex or demanding cognitive operations. Part of the tasks were revised and modified in order to meet the purpose of the proposed study. The levels of complexity for some measures were expanded from the original tasks. For example, the single pair of letter comparisons was increased to a letter comparison task with several letters; and the single pair of patterns comparison task was increased to patterns with 3 patterns comparisons (see APPEDIX A and B).

Two word/non-word lexical decision tasks were also created. The word/non-word lists were generated from the English Lexicon Project Web Site (<http://elexicon.wustl.edu/query13/query13.asp>) at Washington University in St. Louis. The lexical stimuli were generated according to the specified criteria in

Table 2. The words were generated for each easy and difficult condition based on the following factors: word frequency, response times, and mean accuracy. Stimuli across conditions were controlled for word length, neighborhood density and syllable length. Thirty-six words (18 words for the easy condition and 18 words for the difficult condition) and 36 non-words (18 for the easy condition and 18 for the difficult condition) were generated.

The criteria were the same for both words and non-words except for the word frequency factor. For the non-word stimuli, the word frequency factor did not apply. High word frequency (easy version) was set to 300-500, and low word frequency (difficult version) was set to 0-100. Word length (3-6 letters), and number of syllables (two syllables) were the same for both the easy and difficult conditions.

**Table 2. The criteria of word and non-word stimuli with two levels**

Word	Easy	Difficult
Word frequency	High 300-500	Low 0-100
Response times	500-700 ms	800-1000 ms
Mean accuracy	0.8-1	0.5-.07
Word length	3-6 letters	3-6 letters
Orthographic neighborhood size (number)	1	1
Number of syllables	2	2

Non-word	Easy	Difficult
Response times	500-700 ms	800-1000 ms
Mean accuracy	0.8-1	0.5-.07
Word length	3-6 letters	3-6 letters
Orthographic neighborhood size (number)	1	1
Number of syllables	2	2

The sentence completion task was created based on the sentence completion examples from the website (<http://www.englishforeveryone.org/Topics/Sentence-Completion.htm>). A total of 34 items were derived from the grade 2 to the grade 10 items. Table 3 provides the summary of design of this study, and Table 4 and Table 5 present this linguistic task that was selected to approximate the nonlinguistic cognitive processing demands of the RCPM sections.

**Table 3. The summary designs of experiments**

Experimental groups	Research Questions	Independent Variables		Dependent Variable
		Nonlinguistic	Linguistic	
PWA NHC LHD	1. Are there significant differences in the average reaction time among the nonlinguistic 1-3 and linguistic 1-3 tasks among the three participant groups (PWA, LHD, NHC)?  2. Are there significant differences in the degree of slowing between PWA and NHC, between PWA and LHD, and between LHD and NHC?	NL1: CRTT-RT 1 CRTT -RT2 CRTT-RT3 NL2: Pattern-comparison 1 and 2 NL3: RCPM	L1: Letter-comparison 1 and 2 L2: Lexical decision 1 and 2 L3: Sentence completion	Time per stimulus/response (Total time spent in completing correct items for each task /total number of items for each task).
				RTs for research question 2.

**Table 4. The structures of experimental tasks1**

Domains	Nonlinguistic					
Tasks	CRTT-RT-1: Tapping*	CRTT-RT-2*	CRTT-RT-3*	Pattern Comparison 1	Pattern Comparison 2	RCPM
RT-ab format	A	B	B1	C	C	D
	No S → R (10 sec/the number of taps)	S1 (Signal) → R1 (Press a mouse button)	S1 (Signal) → R1 (Move to a Stimulus)	S1 (Same) → R1 S2 (Different) → R2	S1 (Same) → R1 S2 (Different) → R2	S1-S6 → R1 (Press one out of 6 stimul
Assumed Components of RT-ab	Pure Motor {Repetitive Speed}	Perceptual-Motor {Simple Sensory + Simple Motor}	Perceptual-Motor {Simple Sensory + Movement Planning & Simple Execution}	Perceptual-Motor {Simple Perceptual Discrimination & Choice Reaction Time}	Perceptual-Motor {Complex Perceptual Discrimination & Choice Reaction Time}	Perceptual-Motor {More Complex Perceptual & More Complex Choice Reaction Time}
	<ul style="list-style-type: none"> <li>Minimal <u>Attention</u> (task/goal maintenance)</li> </ul>	<ul style="list-style-type: none"> <li><u>Attention</u> (task/goal maintenance)</li> <li><u>Mapping</u> Stimulus to Motor Response</li> </ul>	<ul style="list-style-type: none"> <li><u>Attention</u> (task/goal maintenance)</li> <li><u>Mapping</u> Stimulus to Motor Response</li> <li>Simple <u>Planning</u> Movement Velocities</li> <li><u>Guiding</u> (execution) Movement Velocities</li> </ul>	<ul style="list-style-type: none"> <li><u>Attention</u> (task/goal maintenance)</li> <li><u>Stimulus Comparisons</u> (Discrimination)</li> <li><u>Mapping</u> Stimulus to Motor Response</li> </ul>	<ul style="list-style-type: none"> <li><u>Attention</u> (task/goal maintenance)</li> <li><u>More Stimulus Comparisons</u> (Discrimination)</li> <li><u>Mapping</u> Stimulus to Motor Response</li> </ul>	<ul style="list-style-type: none"> <li><u>Attention</u> (task/goal maintenance)</li> <li><u>More Stimulus Comparisons</u> and more complex visual pattern comparisons</li> <li><u>Reasoning &amp;</u> response selection</li> <li><u>Mapping</u> Stimulus to Motor Response</li> </ul>
Information processing Stages	Early processing	Early processing	Early processing	Early processing	Early processing (Controlled processing)	Early processing Controlled processing
Information Processing Model	⑥	②, ⑥	②, ⑤, ⑥	②, ③, ⑤, ⑥	②, ③, ④-A, ⑤, ⑥	②, ③, ④-B, ⑤, ⑥

**Table 5. The structures of Experimental tasks<sup>2</sup>**

Domains	Linguistic				
Tasks	Letter Comparison 1	Letter Comparison 2	Lexical Decision 1	Lexical Decision 2	Sentence Completion
RT-ab format	C	C	C1	C1	D
	S1 (Same)→R1 S2 (Different)→R2	S1 (Same)→R1 S2 (Different)→R2	S1 (Word)→R1 S2 (Nonword)→R2	S1 (Word)→R1 S2 (Nonword)→R2	S1-S5→R1 (Press one out of 5 stimuli)
Assumed Components of RT-ab	Perceptual-Motor {Simple Perceptual Discrimination & Choice Reaction Time}	Perceptual-Motor {Complex Perceptual Discrimination & Choice Reaction Time}	Perceptual-Motor {Simple Perceptual Discrimination & Choice Reaction Time}	Perceptual-Motor {Complex Perceptual Discrimination & Choice Reaction Time}	Perceptual-Motor {More Complex Perceptual & More Complex Choice Reaction Time}
	<ul style="list-style-type: none"> <li>• <u>Attention</u> (task/goal maintenance)</li> <li>• <u>Comparison</u> (Discrimination)</li> <li>• <u>Mapping</u> Stimulus to Motor Response</li> </ul>	<ul style="list-style-type: none"> <li>• <u>Attention</u> (task/goal maintenance)</li> <li>• <u>More Stimulus Comparisons</u> (Discrimination)</li> <li>• <u>Mapping</u> Stimulus to Motor Response</li> </ul>	<ul style="list-style-type: none"> <li>• <u>Attention</u> (task/goal maintenance)</li> <li>• <u>Word identification</u></li> <li>• <u>Access to lexicon &amp; forming mental representation</u></li> <li>• <u>Comparisons</u> (Discrimination)</li> <li>• <u>Mapping</u> Stimulus to Motor Response</li> </ul>	<ul style="list-style-type: none"> <li>• <u>Attention</u> (task/goal maintenance)</li> <li>• <u>More Stimulus Comparisons</u></li> <li>• <u>Reasoning or logic (restatement, comparison, contrast, cause and effect) &amp; response selection</u></li> <li>• <u>Mapping</u> Stimulus to Motor Response</li> </ul>	<ul style="list-style-type: none"> <li>• <u>Attention</u> (task/goal maintenance)</li> <li>• <u>More Stimulus Comparisons</u></li> <li>• <u>Reasoning or logic (restatement, comparison, contrast, cause and effect) &amp; response selection</u></li> <li>• <u>Mapping</u> Stimulus to Motor Response</li> </ul>
Information processing Stages	Early processing	Early processing (Controlled processing)	Early processing	Early processing Controlled processing	Early processing Controlled processing
Information Processing Model	②, ③, ⑤, ⑥	②, ③, ④-A, ⑤, ⑥	②, ③, ⑤, ⑥	②, ③, ④-A, ⑤, ⑥	②, ③, ④-B, ⑤, ⑥

### 3.3.1 Non-Linguistic Processing Speed Measures

Six non-linguistic processing speed tasks were used in this study. For each task, the number of correct items and reaction or response time were collected to compute the time per item dependent variable.

The finger-tapping task is one of the most common tasks used to investigate human motor function in both normal and pathologic populations (Witt, Laird, & Meyerand, 2008). The tapping task used in this study was the CRTT-RT-1 tapping task and used a mouse button response. In this task, the time interval between taps executed in 10 second was collected, and an average of 3 trials was calculated. The finger-tapping task was used to measure simple motor-related speed.

Two other CRTT tasks measured response times for simple cognitive operations based on perceptual-motor functions. Both are also derived from the reaction time battery of the CRTT-RT. CRTT-RT-2; simple reaction time, measures the time required to respond motorically to each of 30 colored circles or squares appearing in the same location at the center of the screen. Finally, the third CRTT-RT task (CRTT-RT-3) measured reaction time plus movement control time. In CRTT-RT-3, a circle or square (same as the second task) appears at the center of the screen. Participants were instructed to move the cursor from the bottom of the computer screen and click on the token as quickly as possible<sup>19</sup>. Response times (the total time

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<sup>19</sup> In the simple reaction time tasks, the instruction or procedure, “as quickly as possible”, is susceptible to motivational factors, and it could be affected by other factors such as age, gender, education, and even ethnicity (Miller, et al, 1993). Therefore, the instruction for each simple reaction task was provided consistently and uniformly to make the response equally susceptible to those potential factors.

for each item in the task) were collected. The average time per stimulus for 30 trials was used as the dependent measures.

The Raven Coloured Progressive Matrices (CPM A, B, AB forms; Raven, Raven & Court, 1998) consists of a total of 36 items. In this task, each item was presented using E-Prime, and the time for completing each stimulus was collected. For each item, participants were required to determine the missing piece that completes a pattern by pressing a number on the keyboard. The average time per correct item was used as the dependent variable for this task.

The pattern comparison task (Appendix A) consisted of 44 pairs of geometric patterns. Each stimulus was presented by E-Prime and participants were instructed to respond as rapidly as possible, as to whether the two patterns were the same or different. If the patterns were the same, the participant pressed a “1” key on the keyboard. If the patterns were different, they pressed a “2” key on the keyboard. The average time per correct stimulus was served as the dependent variable for this task.

### **3.3.2 Linguistic Processing Speed Measures**

Five linguistic processing speed tasks were initially included in this study. The first language measure was the letter comparison task (Appendix B); a commonly used processing speed task. The letter comparison task required participants to determine, as rapidly and accurately as possible, whether each of 44 pairs of letter strings, consisting of 1 to 3 letters, were the same or different. In the simple reaction time tasks, as with the nonlinguistic tasks, the instruction or procedure, “as quickly as possible”, was susceptible to motivational factors (Miller, et al, 1993). Therefore, the instruction for each linguistic simple reaction task was provided consistently and uniformly to make the response equivalently susceptible to those potential factors. The stimuli



were presented by E-Prime. Both an easy (1) and a difficult (2) version of the tasks were administered. In the easy version, each pair consisted of a single letter. If the letters were the same, the participants pressed a “1” key on the keyboard. If letters were different, the participants press a “2” on the keyboard. In the difficult version of the letter comparison task, each pair of letter strings included 3 letters. The dependent variable was the average time per item for correct responses.

The word/nonword lexical decision tasks were administered to index the lexical level of linguistic processing (Appendix C). The participants made a speeded decision on whether the visual presentation was a word or non-word. All words and non-words were presented via E-Prime. Two demand levels were provided based on the frequency (high and low), and length (short and long) factors. In the easy version, the lists consisted of the words with high frequency and short length; while the difficult version contained words with low frequency and long length. If the stimulus was judged to be a word, participants pressed a “1” key on the keyboard. If the stimulus was judged to be a non-word, participants pressed a “2” key on the keyboard. Instructions to be a fast and accurate as possible were given for each task and the dependent variable was the average time per item for correct responses.

In the sentence completion task, 34 stimulus items were presented using E-Prime. Each item consists of a sentence that had an underlined blank representing a missing word for the sentence and multiple choices for the blank. Participants read the incomplete sentence on the screen and select one of the choices to complete the sentence. Instructions to be as fast and accurate as possible were given for each task and the dependent variable was the average time per item for correct responses.

### **3.4 DATA COLLECTION PROCEDURES**

The basic frames in design and specific structures of the Experiment are summarized in Tables 6-7. Each subject participated in all experimental tasks.

The participants were instructed to complete each task as quickly and accurately as possible. As mentioned earlier, time per item (total completion time for correct items/total number of correct items for each task) was initially collected to compute the dependent variable for data analyses. Subject's data falling at or below chance level for a particular task was excluded from the data analyses. For example, at or below 50% performance was excluded for the binary tasks. For RCPM, performance at or below 18% (6 items out of total 36 items) was excluded for that particular analysis.

All the tasks were presented to each participant in random order, determined by a random number generator (<https://www.random.org/lists/>). Participant recruitment and enrollment were based on participants selected from the Research Participant Registry (<https://www.researchregistry.pitt.edu/Researchers.shtml>), the Western Pennsylvania Patient Registry (WPPR) ([http://www.wppr.pitt.edu/wppr\\_research.html](http://www.wppr.pitt.edu/wppr_research.html)), the Vintage community center, and the Bethel Park community center.

#### **3.4.1 Statistical Analyses**

For the statistical analyses, only correct responses were included. An alpha level of .05 was chosen for all analyses. Normalization procedures such as data trimming and log transformation for RT data were used on the patterns of the data for the data analyses.

The experimental questions addressed are as follows.

1. Are there significant differences in the average time per item among the task means (nonlinguistic 1-3 and linguistic 1-3) and among the three participant groups (PWA, LHD, NHC)?
2. Are there significant differences in the degree of slowing using Brinley Plots between PWA and NHC, between PWA and LHD, and between LHD and NHC?

The dependent variable for each task was time per item, and independent variables were three groups and tasks for the first research question. The number of tasks was determined based on the results of the factor analyses and research purposes after the data collection. Six tasks (3 linguistic and 3 nonlinguistic) were nested within each individual. Two covariates were used as grouping level variables: The group variable was added as the level-1 predictor, and the domain variable (Linguistic and nonlinguistic domains) was added as the level-2 predictor.

A linear mixed model was initially considered and then a generalized linear mixed model was selected for answering the first research question based on the data distribution. A mixed model is a statistical design that includes both fixed effects and random effects. The mixed-model was chosen based on following benefits: 1) Random effects: In a random effects model, both observed and unobserved types of variables can be explained, while a fixed effects model cannot provide information on the segregated effects between observed and unobserved effects (Terhorst, 2007). 2) The mixed model does not require the assumption of independence of subjects, while traditional models such as ordinary least squares (OLS) methods require the assumption of independent subjects (Terhorst, 2007). 3) Cross effects: A mixed model can provide information on both subject groups and items simultaneously. 4) Mixed models provide a better approach than ANOVA and OLS methods in terms of treating missing data, treating

continuous and categorical responses, and modeling heteroskedasticity and non-spherical error variance (Baayen et al., 2008).

Groups (PWA, LHD, and NHC) and domains (linguistic and nonlinguistic domains) were counted as fixed factors and subjects were counted as a random factor in the model. Thus, several different models were built based on the multiple combinations of fixed and random factors, and best fits were determined. The best-fit model was chosen for the data analyses. In order to compare the goodness of fit of multiple models, the values of deviance, a quality-of-fit statistic for a model, were compared among the models, and Akaike's Information Criterion (AIC) and Schwarz's Bayesian Criterion (SBC) were used. The model that produced larger AIC and SBC values was considered as the best-fit model (Singer, 1998).

The following assumptions for the mixed model were checked before the statistical data analyses for the research questions: 1) The fixed and random parts of the model contained the right variables. 2) The fixed part of the model residuals were determined to be normally distributed and 3) have constant variance. 4) The random part of the model residuals were normally distributed (Terhorst, 2007).

According to the first research question, the main effect of groups, the main effect of domains, and the interaction effects between the groups and domains were computed. For these statistical analyses, SAS PROC MIXED statistical programming packages were utilized.

In this study, Brinley plots, used in previous general slowing studies, were generated to compare the degree of slowing by computing the raw mean time data for groups (PWA, LHD and NHC), and by plotting the mean values against each other. As such, times for all experimental tasks data were plotted using linear regression between PWA and NHC, between PWA and LHD, and between LHD and NHC on the tasks. The estimate of the degree of slowing

was based on the slope of the regression lines. The generated regression equations (slopes) and the plots provide the information on the overall group differences in slowing. For the computation of regression analyses, the IBM SPSS statistics package was used.

## **4.0 RESULTS**

### **4.1 DATA ANALYSES & RESULTS**

#### **4.1.1 Task Selection Process: Principal Component Analysis**

Eleven tasks were used for the data collection, and 6 tasks (3 linguistic and 3 nonlinguistic tasks) out of the 11 tasks were chosen based on the Principal component analysis (PCA). The PCA, with varimax rotation, was performed for the 11 tasks in order to identify those tasks with the greatest shared variance. Table 6 summarizes the coefficients for these three components. The data for the PCA were based on the raw RT values from the PWA and the LHD groups because these two brain damaged groups' reaction times were significantly different from the NHC group, and the PWA and LHD groups were, based on the results of Brinley plots, differed in the degree of slowing.

The criteria for choosing the tasks based on the PCA were as follows: 1) the tasks with the highest or second highest value were chosen in each factor, and 2) the tasks that were not highly loaded in other factors (PA-1 and PA-2 were the exceptional cases for this criteria and the reason for its exceptionality is as follows) were chosen.

**Table 6. Coefficients derived from the PCA Rotated Component Matrix**

	Factor 1	Factor 2	Factor 3
PA-1	.877	.226	.034
PA-2	.815	.164	.007
RCPM	.598	.564	.103
LX-1	<b>.863</b>	.192	.173
LX-2	.778	.357	-.054
SC	.625	.309	.228
CRTT-RT1	-.033	<b>.629</b>	.556
CRTT-RT2	.245	.017	<b>.891</b>
CRTT-RT3	.262	<b>.766</b>	-.042
LE-1	<b>.880</b>	.188	.318
LE-2	<b>.861</b>	-.116	.341

**Coefficients bolded are those selected to represent each factor.**

PA1=Pattern Comparison 1; PA2=Pattern Comparison 2; RCPM: Raven's Coloured Progressive Matrices; LE1=Letter Comparison 1; LE2=Letter Comparison 2; LX1=Lexical Decision 1; LX2=Lexical Decision2; SC=Sentence Completion

Letter comparison 1 and 2, and Lexical decision 1 were identified as the first component. The nonlinguistic Pattern Comparison 1 and 2 tasks were identified with linguistic tasks in the first factor; however, those were not selected to represent the linguistic domain because it was speculated that they loaded in the first factor due to linguistic representation or verbal mediation in the response format<sup>20</sup>.

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<sup>20</sup> The required response format was linguistic (same or different) across those highly loaded tasks: PA-1 and 2, LX-1 and 2, LE-1 and 2. Therefore, it is likely to be mediated verbally or at least linguistically represented implicitly.

CRTT-RT1 (motor speed) and CRTT-RT3 (movement control) were identified, as a second component. CRTT-RT2 (simple reaction time) was selected as a third component. Therefore, CRTT-RT-1, CRTT-RT-2, and CRTT-RT-3 were chosen as nonlinguistic tasks. Letter comparison 1 and 2, and Lexical decision 1 were selected as linguistic tasks for addressing the first research question. The final selection of the experimental tasks was bolded in the Table 6.

#### **4.1.2 Data Processing For the GLMM Analyses**

The original data were log-transformed because the data were not normally distributed based on the histogram and the Kolmogorov-Smirnov normality test. After log-transformation, the data distribution remained skewed. Therefore, a Generalized Linear Mixed Model (GLMM) was selected for analysis because the GLMM is known as a flexible model for skewed distributions (Vock et al, 2012; Malehi et al, 2015). Outliers (3SD) were excluded from each individual data for the data analyses.

#### **4.1.3 Statistical Models**

Four different statistical models were generated in order to compare predictor variances. The first model was an empty model without a predictor. The second model had group as a predictor. Model three had domain as a predictor. Model four included domain\*group interaction as well as both domain and group as predictors. Therefore, model four represented the complete model with which to answer the first research question. This model is represented as follows.



$$\text{Mixed Model: } (\gamma_{00} + \gamma_{01}W_j + u_{0j}) + (\gamma_{10} + \gamma_{01}W_j + u_{1j}) * I(X=2) + (\gamma_{20} + \gamma_{21}W_j + u_{2j}) * I(X=3) + \varepsilon_{ijl}$$

Hierarchical Linear Model:

$$\text{Level 1: } Y_{ijl} = \beta_{0j} + \beta_{1j}X_i + \varepsilon_{ijl}$$

$$\text{Level 2: } \beta_{0j} = \gamma_{00} + \gamma_{01}W_j + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{01}W_j + u_{1j}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}W_j + u_{2j}$$

In this model, the level-1 predictor was group (where  $X_i$ ,  $i$  is the index of group), and the level-2 predictor was domain (where  $W_j$ ,  $j$  is the index of domain).  $\varepsilon_{ijl}$  is the random error associated with the measurement in domain  $j$  on the  $l^{\text{th}}$  subject that is group  $i$ .  $u_{0j}$  and  $u_{1j}$  are level-2 residuals. At the second level,  $\beta_0$  is the overall effect of group 1, and  $\beta_0 + \beta_{1j}$  is the overall effect of group 2 and the difference between the group 1 and 2.  $\beta_0 + \beta_{2j}$  is the overall effect of group 3 and the difference between groups 1 and 3.

The gamma distribution was selected for the model because all the values are positive and positively skewed in the distribution with a log link function. The log link function is as follows:

$$Y_{ijl} = u_{ij} + \varepsilon_{ijl}$$

$$u_{1j} \sim \Gamma(W_{ij})$$

$$g(W_{ij}) = -\frac{1}{W_{ij}} = \alpha_i + \beta_j + (\alpha\beta)_{ij}$$

$Y_{ijl}$  is the RTs in domain  $j$  on the  $l^{\text{th}}$  subject that is in group  $i$ , and  $u_{ij}$  is the overall effect

of group and domain.  $u_{ij} \sim \Gamma(W_{ij})$  means  $u_{ij}$  is a gamma distribution.  $W_{ij}$  is a parameter of the gamma distribution.  $g$  is link function, so  $g(W_{ij})$  is the link function of the gamma distribution.  $\alpha_i$  is the effect of group, and  $\beta_j$  is the effect of domain.  $(\alpha\beta)_{ij}$  is the interaction effect of group and domain.

Each of the four models was computed with three different covariance structures (Unstructured, Autoregressive, and Compound Symmetry) using SAS statistical package 23, PROC GLIMMIX. However, the variance of the estimate of the covariance parameter was zero across all models. Therefore, the random effect of subject was removed based on the SAS online guideline in the official SAS website: ([https://support.sas.com/documentation/cdl/en/statug/63962/HTML/default/viewer.htm#statug\\_mixed\\_sect031.htm](https://support.sas.com/documentation/cdl/en/statug/63962/HTML/default/viewer.htm#statug_mixed_sect031.htm)), and the models were computed again and this result was selected as the final solution.

#### **4.1.4 Results for the First Research Question**

The summary of fit statistics and other comparable values in the Model 1-4 are as shown in Table 7. Model 1 was the empty model without a predictor, and it was generated to compare with other models with predictors. Group was selected as a predictor for Model 2, where RTs were significantly different among groups ( $F(2,357) = 6.36, p = .0019$ ). With the Bonferroni adjustment for the multiple comparisons, the PWA group RTs was not significantly different from the LHD group ( $t = 1.43, p > .05$ ), while the PWA group was significantly different from the NHC group ( $t = 3.49, p = .0005$ ). The LHD group was not significantly different from the NHC group ( $t = 1.84, p > .05$ ) in the Model 2. Selecting domain as the predictor for Model 3, significant

RT differences ( $F(2,358) = 82.93$ ,  $p < .0001$ ) were found with the nonlinguistic domain being significantly shorter than the RTs in the linguistic domain ( $t = -9.11$ ,  $p < .0001$ ).

**Table 7. Summary of results for Models 1 through 4.**

	Model 1	Model 2: group	Model 3: domain	Model 4: group, domain, group*domain
-2Log Likelihood	252.31	239.78	177.69	160.53
AIC	252.31	247.78	183.69	174.53
Person Chi-Square	4.99	4.78	4.34	4.13
<i>F</i> value of predictors		group: 6.36**	domain: 82.93****	group: 7.60** domain: 97.10**** group*domain: 1.15

\*\*  $p < .01$ , \*\*\*\*  $p < .0001$

In order to address the first research question, the main effects of group and domain, and the interaction between the groups and domains were computed (Model 4). There were significant main effects for group, ( $F(2,354) = 7.60$ ,  $p = .0006$ ) and domain ( $F(1,354) = 84.84$ ,  $p < .0004$ ). There was no significant interaction between group and domain ( $F(2,354) = 1.15$ ,  $p > .05$ ). Post hoc analyses were computed with a Bonferroni adjustment computed for the multiple comparisons. These analyses revealed that the two brain-damaged groups were significantly different from the NHC group in the Model 4 (PWA-NHC,  $t = 3.81$ ,  $p = .0002$ , LHD-NHC,  $t = 2.03$ ,  $p < .05$ ), however the PWA group was not significantly different from the LHD group ( $t = 1.55$ ,  $p > .05$ ). The nonlinguistic RT tasks were significantly ( $t = -9.21$ ,  $p < .0001$ ) shorter than the RTs for the linguistic tasks.

#### 4.1.5 Results for the Second Research Question: Degree of Slowing using Brinley Plots

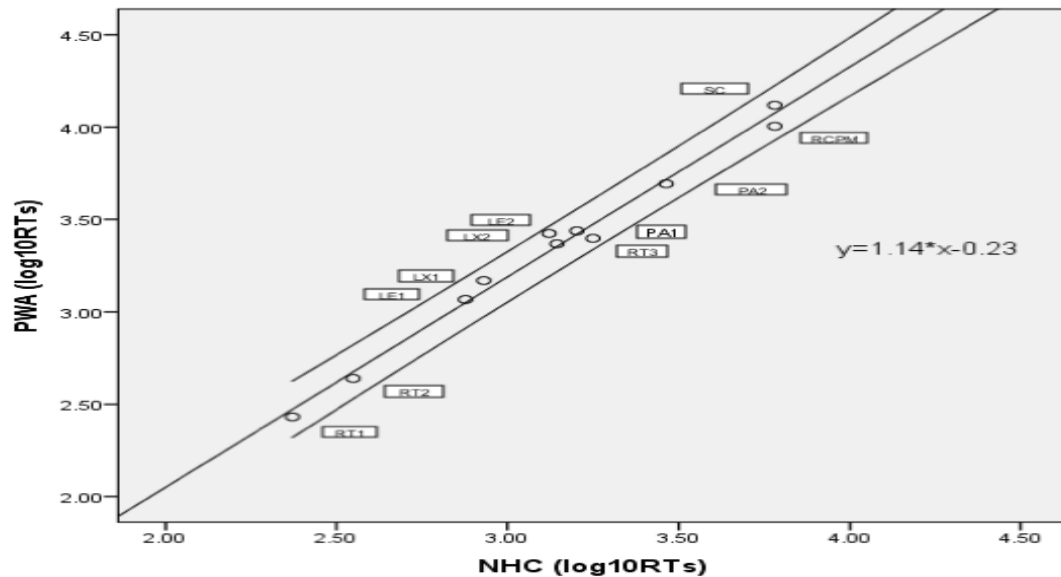
In order to examine the degree of slowing to address the second research question, a regression was computed using the average log-transformed RT scores for each of the 11 tasks. Table 8 summarizes the means and standard deviations of the raw RTs for all tasks for each group. These data are plotted in Figure 6 using a Brinley Plot, contrasting each group.

**Table 8. Average and Standard Deviation (SD) reaction times (RT) for each of the experimental tasks for each of the participant groups.**

TASKS	PWA	LHD	NHC
	Average RT and (SD)	Average RT and (SD)	Average RT and (SD)
CRTT-RT1	270.68 (32.87)	274.64 (28.42)	235.17 (13.59)
CRTT-RT2	437.26 (120.81)	412.10 (129.71)	354.74 (69.65)
CRTT-RT3	2495.31 (867.35)	2125.86 (693.60)	1776.97 (668.50)
PA1	2739.05 (1172.85)	2040.19 (812.90)	1594.23 (546.16)
PA2	4925.68 (2292.58)	3880.12 (1854.60)	2905.24 (1523.04)
RCPM	10122.36 (5059.64)	7923.63 (4344.27)	6026.79 (2789.34)
LE1	1167.09 (367.21)	935.71 (231.02)	754.45 (140.98)
LE2	2652.87 (773.34)	1772.14 (528.16)	1323.55 (325.87)
LX1	1476.12 (478.18)	1134.80 (367.57)	853.29 (187.03)
LX2	2332.18 (1017.10)	1861.94 (841.33)	1394.36 (555.84)
SC	13155.41 (10832.00)	9378.97 (8505.21)	6023.28 (5463.10)

PWA=People with aphasia; LHD=Left Hemisphere Damaged people without aphasia; NHC= Normal Healthy Controls; PA1=Pattern Comparison 1; PA2=Pattern Comparison 2; RCPM: Raven's Coloured Progressive Matrices; LE1=Letter Comparison 1; LE2=Letter Comparison 2; LX1=Lexical Decision 1; LX2=Lexical Decision2; SC=Sentence Completion

The log-transformed RTs for all tasks for the PWA, compared with the NHC group, yielded a significant simple linear regression ( $F(1, 9)=780.223$ ,  $p<0.001$ ), with a slope of 1.14 and a correlation of 0.989 ( $R^2$ ) (Figure 6). This provides evidence that the PWA group performed 1.14 times slower than the NHC group and all tasks were relatively equivalently slowed as evidenced by their shared common regression line and the values falling within the 95% CI around the slope.

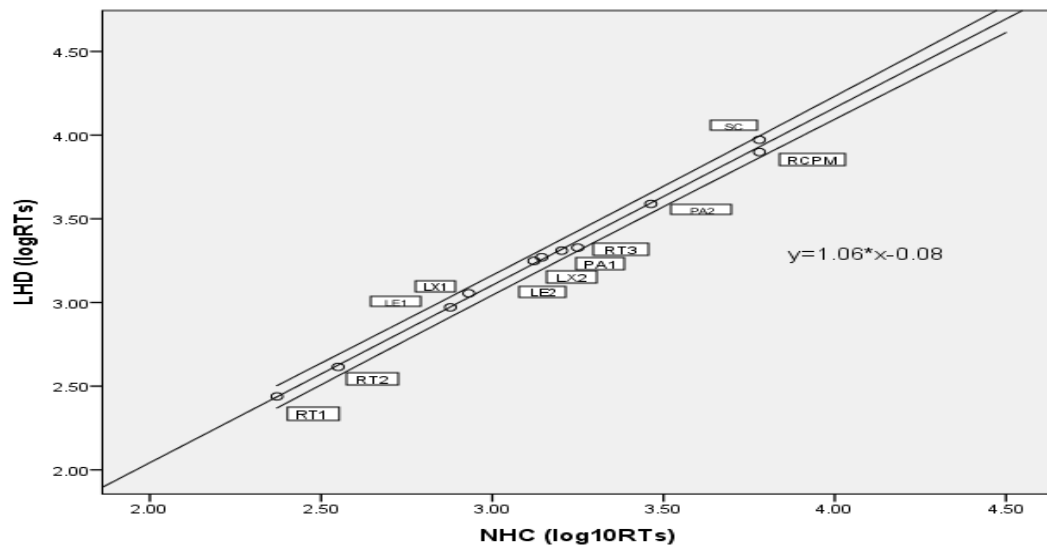


PWA=People with aphasia; LHD=Left Hemisphere Damaged people without aphasia; NHC= Normal Healthy Controls; PA1=Pattern Comparison 1; PA2=Pattern Comparison 2; RCPM: Raven's Coloured Progressive Matrices; LE1=Letter Comparison 1; LE2=Letter Comparison 2; LX1=Lexical Decision 1; LX2=Lexical Decision2; SC=Sentence Completion

**Figure 6. Brinley plot of RTs across 11 tasks for PWA and NHC**

The log-transformed RTs for all tasks across the LHD and NHC groups yielded a significant simple linear regression ( $F(1,9)=3544.967$ ,  $p<0.001$ ), with a slope of 1.06 and a correlation of 0.997 ( $R^2$ ) (Figure 7). This provides evidence that the LHD group performed 1.06

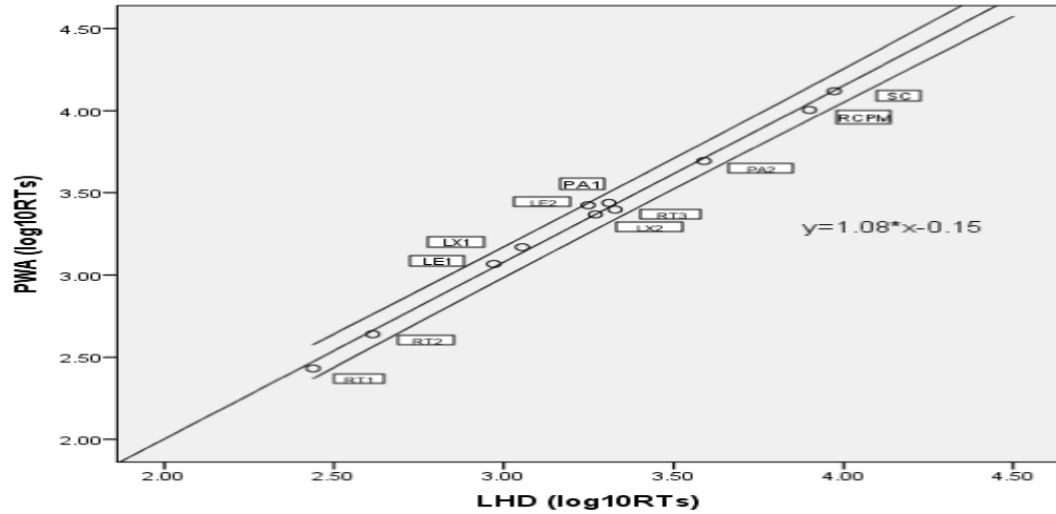
times slower than the NHC group and all tasks were relatively equivalently slowed as evidenced by their shared common regression line and the values falling the 95% CI around the slope.



PWA=People with aphasia; LHD=Left Hemisphere Damaged people without aphasia; NHC= Normal Healthy Controls; RT1=CRTT-RT1; RT2=CRTT-RT2; RT3=CRTT-RT3; PA1=Pattern Comparison 1; PA2=Pattern Comparison 2; RCPM: Raven's Coloured Progressive Matrices; LE1=Letter Comparison 1; LE2=Letter Comparison 2; LX1=Lexical Decision 1; LX2=Lexical Decision2; SC=Sentence Completion

**Figure 7. Brinley plot of RTs across 11 tasks for LHD and NHC**

The log-transformed RTs for all tasks across the PWA and LHD groups yielded a significant simple linear regression ( $F(1, 9)=1715.8$ ,  $p<0.001$ ), with a slope of 1.08 and a correlation of .995 ( $R^2$ ) (Figure 8). This also provides evidence that the PWA group performed 1.08 times slower than the LHD group and all tasks were relatively equivalently slowed as evidenced by their shared common regression line and the values falling within the 95% CI around the slope.



PWA=People with aphasia; LHD=Left Hemisphere Damaged people without aphasia; NHC= Normal Healthy Controls; RT1=CRTT-RT1; RT2=CRTT-RT2; RT3=CRTT-RT3; PA1=Pattern Comparison 1; PA2=Pattern Comparison 2; RCPM: Raven's Coloured Progressive Matrices; LE1=Letter Comparison 1; LE2=Letter Comparison 2; LX1=Lexical Decision 1; LX2=Lexical Decision2; SC=Sentence Completion

**Figure 8. Brinley plot of RTs across 11 tasks for PWA and LHD**

#### 4.1.6 Additional Analyses

Several additional analyses were performed using SPSS to clarify the subsequent questions more specifically related to aphasia specificity and domain specificity. First, log-transformed RTs for the two pathological groups were computed across all the tasks using the non-parametric Mann-Whitney U test in order to investigate whether the PWA group performed significantly differently from the LHD group in the tasks requiring higher order cognitive computation. As summarized in Table 9, the PWA and LHD were significantly different in the most difficult nonlinguistic and linguistic tasks. That is, the PWA group was significantly slower than the LHD group for the Raven Coloured Progressive Matrices ( $Mdn=18.40$  for PWA,  $Mdn=12.60$ ,  $U=69$ ,  $z=-1.804$ ,  $p<.05$ ) and for the Sentence Completion task ( $Mdn=18.67$  for PWA,  $Mdn=12.33$ ,  $U=65$ ,  $z=-1.970$ ,  $p<.025$ ).

**Table 9. Between PWA and LHD group differences derived from the Mann-Whitney U, with Z scores and significance values for the 11 tasks depicted in the Brinley Plots**

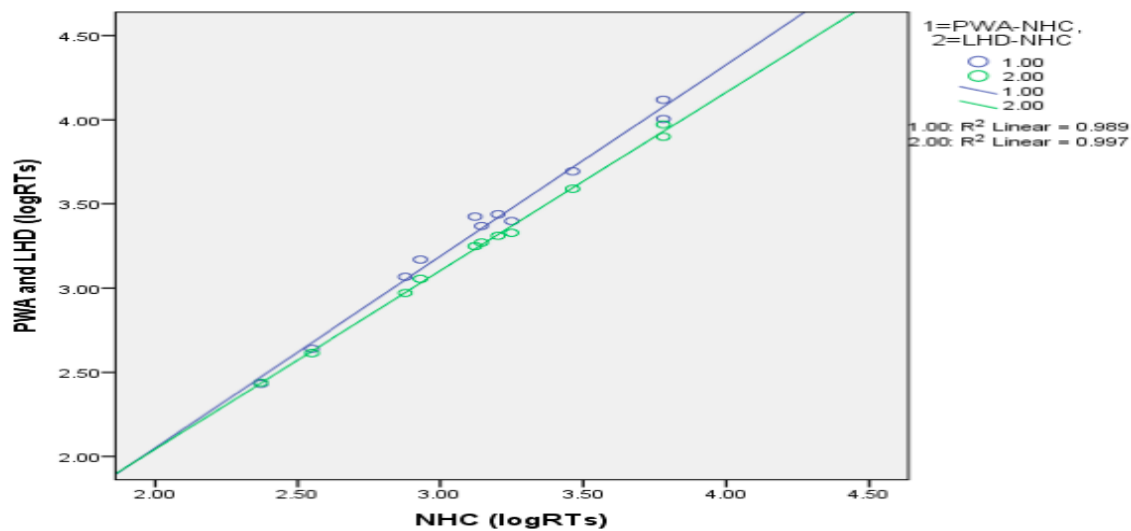
	RT1	RT2	RT3	PA1	PA2	RCPM	LE1	LE2	LX1	LX2	SC
U	108	95	79	50	63	69	66	51	59	72	65
Z	-.19	-.73	-1.39	-2.59	-2.05	-1.80	-1.93	-2.55	-2.22	-1.68	-1.97
Sig.	.44	.24	.87	.01**	.02*	.04*	.03*	.01**	.01*	.05*	.025*

\*  $p < .05$ ; \*\*  $p < .01$ . PWA=People with aphasia; LHD=Left Hemisphere Damaged people without aphasia; NHC=Normal Healthy Controls; RT1=CRTT-RT1; RT2=CRTT-RT2; RT3=CRTT-RT3; PA1=Pattern Comparison 1; PA2=Pattern Comparison 2; RCPM: Raven's Coloured Progressive Matrices; LE1=Letter Comparison 1; LE2=Letter Comparison 2; LX1=Lexical Decision 1; LX2=Lexical Decision2; SC=Sentence Completion

In addition, domain specificity was tested within the PWA group. The results were based on the log-transformed data of the 6 tasks selected from the PCA analysis. There was no significant difference between the nonlinguistic ( $Mdn=2.67$ ) and linguistic ( $Mdn=4.33$ ) domains ( $U=2$ ,  $z=-1.091$ ,  $p>.05$ ) on these six tasks.

Differences between the two brain-damaged groups, relative to the NHC group, were also plotted in order to highlight the differences among the lower and higher cognitive demand tasks requiring less and more complex cognitive computations. Brinley plots between the PWA and the NHC, and between the LHD and the NHC were plotted. The regression lines for these two groups diverged (Figure 9) on the higher order tasks.





**Figure 9. Brinley plots of RTs between PWA-NHC and LHD-NHC for the 11 experimental tasks depicted in figures 6, 7 and 8.**

The Brinley plots were generated with the averaged raw RT data as well as with the log-transformed data in Appendix L-O for better illustrative purposes. Therefore, the patterns of the results were comparable.

## **5.0 DISCUSSION & CONCLUSION**

The primary purpose of this study was to investigate the nature of slowing in PWA and to compare their performance to the LHD and NHC groups on linguistic and nonlinguistic tasks selected to represent a range of cognitive task demands. More specifically, the study was directed toward examining aphasia specificity and domain specificity. Reaction and response times were compared to examine the differences in processing speed performance among those groups and also plotted to investigate whether the groups differed in the degree of slowing if present.

The first research question focused on differences in reaction times across participant groups and by cognitive domain. The results of the mixed effects model revealed significant main effects of groups and domains, and no significant interactions among groups, or domains. Specifically, the two brain-damaged groups (PWA and LHD) produced significantly longer reaction times across tasks than the NHC group. The two brain-damaged groups' reaction times were significantly different from each other from the simple perceptual tasks to the higher order tasks except for the sensory-motor tasks, CRTT-RT1-3.

These results revealed that both the brain-damaged groups evidenced slowing across domains (domain (non)specificity) and the aphasic group evidenced additional slowing relative to the LHD group (aphasic (non)specificity). The aphasia specific hypothesis proposed that the PWA group would produce reaction times that would be significantly longer than the other two groups. The finding was in fact consistent with the hypothesis except for the results from the perceptual-motor-related tasks (CRTT-RT 1-3). Based on the aphasia specificity hypotheses C, where both the PWA and LHD groups were predicted to perform nonsignificantly different

from each other, the result of no difference in the motor-perceptual tasks is interpreted as no aphasia specificity. However, the two brain-damaged groups performed significantly differently from each other in the remaining linguistic and nonlinguistic tasks, which would be interpreted as aphasia specific slowing; specifically in the higher-order cognitive, more cognitively demanding tasks. Therefore, the results of the main effect of group and the additional analyses on the two brain damaged groups are consistent with the interpretation that the PWA group's slowing is related to both the left-hemisphere brain damage as well as the presence of aphasia. Analogous to the general slowing hypothesis associated with aging (Cerella, 1980; 1985), this pattern is consistent with a brain-damaged related slowing. Therefore, the general slowing hypothesis (Cerella, 1980; 1985) might be expanded to include a brain-damaged component as well as one of aging. "General slowing" here can be interpreted as the slowing specifically related to the brain-damaged conditions secondary to stroke, not from the aging process because any aging effect was assumed to have been controlled by the comparable age of participants across the groups. These results are consistent with an interpretation that a brain-damage component might be considered as an additional mechanism for the slowing that is additive to and accompanies the process of aging.

The types or the complexity of the tasks may explain the lack of difference between the two brain-damaged groups. The 6 tasks that were selected for inclusion in the mixed model from the eleven total tasks, was based on the PCA analysis. These 6 tasks required relatively low levels of cognitive computation as the cognitively higher order tasks were excluded based on the PCA. Therefore, the results showing no difference in the two groups, based on this analysis, are limited to the cognitively low demand tasks. This is supported by an examination of the Brinley plots (Figure 9) where the two brain-damaged groups exhibited a divergent

pattern of slowing for the higher order/greater demand tasks that require more complex cognitive computations.

As previously mentioned, there was no interaction between group and domain, indicating no differential effects of domain on group. Domain specificity based on the 6 tasks was not supported for the PWA group. That is, the PWA group did not perform significantly slower on the linguistic tasks than on the nonlinguistic tasks. However, while the RTs were essentially the same for RCPM (*Mean*: 6026.79, *SD*: 935.05) and SC (*Mean*: 6023.28, *SD*: 5463.1) for the NHC group, the PWA group was significantly slower in the SC (*Mean*: 13155.41, *SD*: 4047.01) than in the RCPM (*Mean*: 10122.36, *SD*: 5059). Interestingly, supplementary testing revealed that like the PWA group performance, the RCPM time (*Mean*: 7923.63, *SD*: 4344.27) was also significantly shorter than the SC time (*Mean*: 9378.97, *SD*: 8505.21) for the LHD group. These times that were intermediate between the PWA and the NHC groups but with longer RTs on the most difficult language task is interpreted as evidence that the LHD group's pattern of performance is similar to that of the PWA. This interpretation challenges the utility of the PCA analysis for identifying factors that can be interpreted as linguistic versus nonlinguistic. It is speculated that the identified components are related to the difficulty of the tasks, rather than the underlying nature of the mental representations that were predetermined as linguistic and nonlinguistic. The resolution of this important issue will require additional research beyond the current study.

In order to address the second question concerning the degree of slowing, Brinley plots were constructed for the 11 tasks contrasting groups. According to these results, a linear slowing across all experimental tasks was evident for each of the two group comparisons (between PWA and LHD, between PWA and NHC, and between LHD and NHC) using simple linear

regression. The PWA group's performance was 2 times slower than NHC participants, while the LHD group was 1.46 time slower than the NHC participants. Importantly, the PWA group was 1.4 times slower than the LHD group who presented without evidence of aphasia according to the diagnostic criteria used in this study. These findings support the general slowing hypothesis (Cerella, 1980) based on the result from the two brain-damaged groups relative to the NHC group, even though the original slowing hypothesis is based on aging, not brain damage. However, the brain-damaged groups leave the domain specificity question substantively unanswered. That is, as discussed above, both brain-damaged groups demonstrated significantly slower performance on the most difficult language task (SC), compared to the most difficult nonlinguistic task (RCPM). There is, however, no way to equate these tasks for overall cognitive demand or processing difficulty, leaving this interpretation of the findings speculative.

Several critical issues have to be discussed relative to the results from both the Brinley plots and mixed effects model. First, both groups of left-hemisphere brain-damaged participants evidenced slowing across all tasks compared to the NHC. The LHD group performed like the PWA group in that they differed only in the degree of slowing. Therefore, it is hypothesized that there is a brain-damaged related slowing. Many of the LHD group (12 out of 15) had never received a diagnosis of aphasia. However, as discussed above, this may be the result of inadequate assessment or insensitive measures of aphasia at the time of their stroke. There is the possibility that the diagnostic was simply inadequate. It is speculated that if they had received a comprehensive language evaluation, they too might have met the criteria for being aphasic immediately after their stroke. In other words, the problem might be in the lack of sensitivity of the assessment and that they were actually "sub-clinically" aphasic.

One possible explanation for the observed brain-damaged related slowing in the LHD group is the likelihood of reduced brain connectivity after the stroke. Reduced brain connectivity can reduce processing efficiency for the tasks used in this study, compared to the NHC group's function. While the LHD group did not evidence aphasia, perhaps they were subclinically aphasic and didn't display sufficient signs or severity of signs to be diagnosed as such. Several recent studies have reported less effective connectivity in stroke survivors than controls (Baldassarre et al, 2016; Li et al, 2014; Rehme & Grefkes, 2013). However, none of these studies specified whether the stroke survivors had aphasia or not. Therefore, we cannot be sure whether the lack of connectivity is due to the brain-damaged related symptom or the sub-clinical aphasics' symptom without testing language with tasks of sufficient complexity effects to reveal deficits, relative to control participant's performance, if present.

Another issue involves the differences between the PWA and the LHD participants, assessing aphasia-specific slowing. If language-specific slowing exists, and that is the core of the difference between the two groups, the PWA group were required to exhibit better performance on the nonlinguistic tasks, and dominantly slowed RTs in the linguistic tasks. The RTs from both groups were not different on the CRTT-RT 1-3 tasks, which primarily engage nonlinguistic sensorimotor functions. While the perceptual-sensorimotor tasks were not performed differently between the PWA and LHD groups, the cognitively demanding (low to high) tasks were different regardless of domains. Therefore, the difference between the two groups might begin even at relatively low-level cognitive tasks regardless of the domains of the tasks. Again, the difference between the PWA and the LHD groups was larger on the most difficult tasks based on the diverging pattern.

If the above interpretation is accurate, the definition of aphasia would not be limited to deficits within the language domain. This notion is consistent with the formal definition of aphasia of McNeil and Pratt (2001) who proposed that aphasia is inordinately a language deficit but not exclusively or selectively a disorder limited to the language domain. However, based on this study it is too early to draw a firm conclusion about the definition or diagnostic criteria for aphasia. Future studies will clarify aphasia specific slowing vs. brain-damaged slowing. Villard & Kiran's (2016) review is also consistent with the domain-general deficits in aphasia. They suggested domain-general cognitive resources, specifically attention, as a supporting cognitive mechanism for language processing in PWA. Therefore, the possibility of domain-general deficits in PWA might be plausible, even though the domain-general cognitive resources they deal with is attention, not processing speed per se.

The patterns of the results are consistent with a cognitive complexity account rather than a linguistic versus nonlinguistic-specific domain account. The CRTT-RT tasks 1-3 showing no difference between the PWA and the LHD groups require minimal cognitive computation relative to the rest of the tasks. Therefore, cognitive complexity might be the source of the difference between the two brain-damaged groups.

This speculation of the cognitive complexity is consistent with the complexity hypothesis from the aging general slowing hypothesis (Cerella, 1980). According to Cerella (1980), the degree of slowing was steepest for the complex tasks (1.62) requiring more/higher order processing compared to the sensorimotor tasks (1.14) similar to those employed in the current investigation that imposed less task demands. CRTT-RT tasks 1-3 represent sensorimotor slowing relatively well, and the higher order tasks such as the RCPM and SC tasks also represent more demanding processing tasks.

Another explanation for the differences between the PWA and LHD groups might be the lesion size. Several studies have shown that the size of lesion was related to the poor performance of PWA or the severity of aphasia as a recovery factor (Sandberg, 2017, Agis et al, 2016). Sandberg (2017) tested hypoconnectivity of resting-state networks in PWA and found that the lesion size and WAB AQ were negatively correlated ( $r = -0.64$ ,  $p < 0.01$ ). Agis et al (2016) found that lesion size was negatively correlated ( $r = -0.59$ ;  $p = 0.003$ ) with the total content units from the analysis of the Cookie Theft Picture in people with LH stroke.

According to Turken et al (2008), cognitive processing speed on the Digit-Symbol test was associated with white matter lesions, including superior longitudinal fasciculus (SLF), in left-hemisphere stroke patients (presence of aphasia was not reported) using diffusion tensor imaging (DTI). They found that voxel-based lesion-symptom mapping analysis (VLSM) revealed regions that overlapped with the SLF in the left parietal white matter. If the SLF is critical for processing speed, it would be important to determine if this lesion site (and size) was involved for PWA. Considering that SLF covers an extensive area in the brain<sup>21</sup>, it is possible that both having a larger lesion and several different lesion sites within the SLF could be related to slow processing speed in PWA.

This study was not designed to investigate either lesion size or location<sup>22</sup>. Nonetheless,

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<sup>21</sup> Association fibers from the SLF are connected to the frontal, occipital, parietal, and temporal lobes. According to Makris et al (2005), “SLF I is located in the white matter of the superior parietal and superior frontal lobes and extends to the dorsal premotor and dorsolateral prefrontal regions. SLF II occupies the central core of the white matter above the insula. It extends from the angular gyrus to the caudal--lateral prefrontal regions. SLF III is situated in the white matter of the parietal and frontal opercula and extends from the supramarginal gyrus to the ventral premotor and prefrontal regions. The fourth subdivision of the SLF, the arcuate fascicle, stems from the caudal part of the superior temporal gyrus arches around the caudal end of the Sylvian fissure and extends to the lateral prefrontal cortex along with the SLF II fibers.”(p. 854)

<sup>22</sup> Brain imaging scans for many of PWA and LHD participants were not available to quantify the lesion site and size.



based on the lesion descriptions from clinical reports for both PWA and LHD participants in this study might provide some insight into the results of this study. There were, however, disparate descriptions of the lesions. For example, seven out of fifteen LHD participants were described as having “small”, “patchy”, “focal” areas, and “tiny foci” for their lesions. Four of fifteen participants from the PWA group were described as “moderately large”, and “large areas” for the lesions. Assuming that the PWA group had larger lesions than the LHD group, the additional slowing for the PWA group relative to the LHD groups might be due to the lesion size. Additionally, it is possible that larger lesions might include more critical areas for language processing networks.

There are also cases that don’t fit the generalizations about the lesion size and site from the selected subjects discussed above. Participant LHD 1 showed a very large lesion in her scan (see Figure 10<sup>23</sup> upper image), but she evidenced no speech and language deficits subsequent to a stroke. Contrarily, participant PWA 15 evidenced a lesion (see Figure 10 lower image) that was smaller than the LHD participant, however, she displayed prominent and persistent aphasia symptoms following a stroke. These sources of alternative evidence for a lesion size account explaining the difference between the PWA and the LHD groups demands further prospective studies and more finite lesion analyses.

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<sup>23</sup> Lesions were traced manually using ITK-SNAP software on the high-resolution MPRAGE images that were collected.

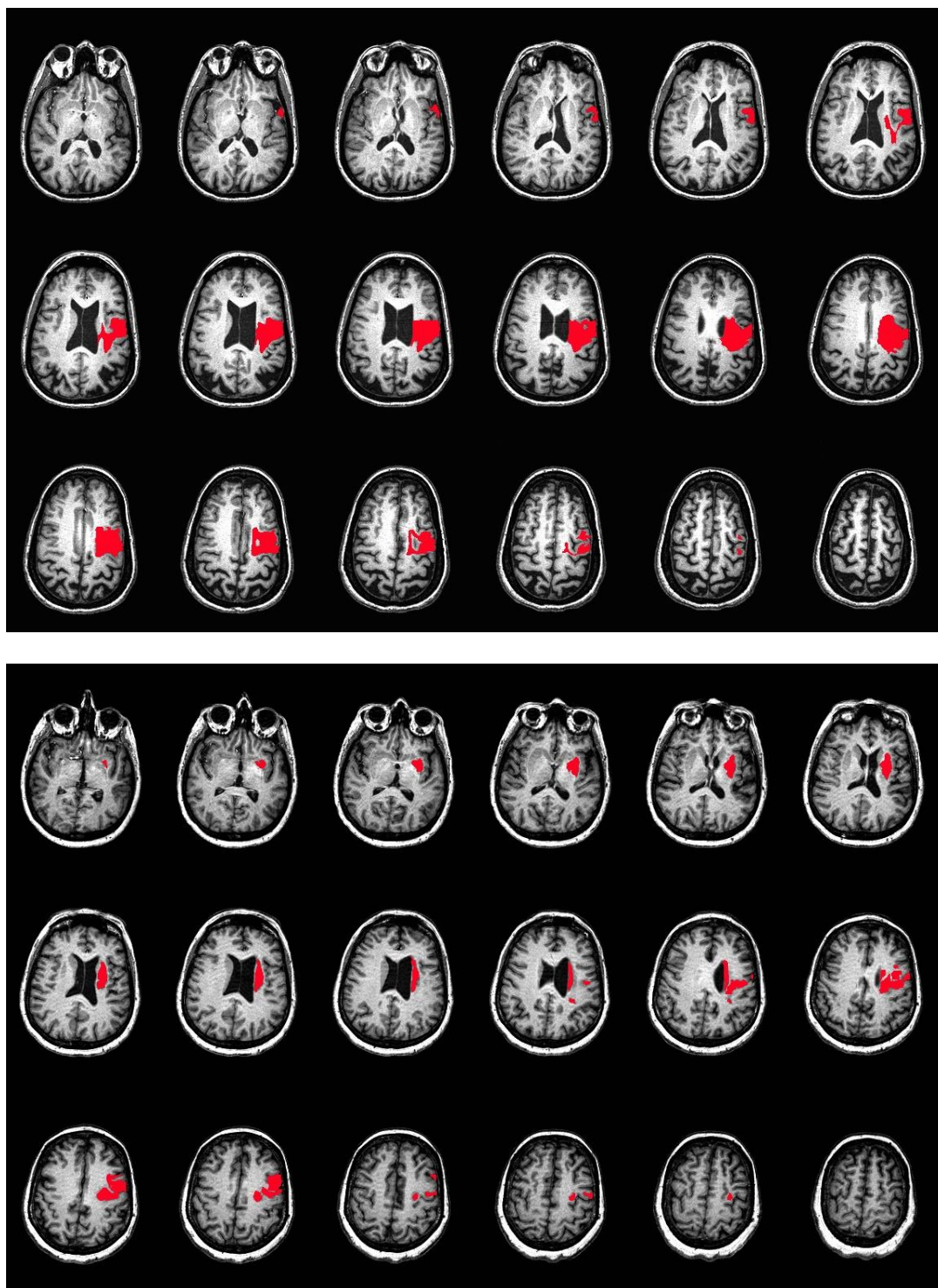


Figure 10. Lesion-traced fMRI snapshots of LHD 1 (upper) and PWA 15 (lower)

The following conclusions can be drawn from this study. Processing speed on perceptual, sensorimotor (or perceptual-motor), and higher order cognitive tasks is critical for the full description and differentiation of the PWA from individuals with left hemisphere lesions that present without aphasia as well as from age-matched normal healthy controls. Specifically, higher order cognitive tasks might serve an important role in differentiating the groups given the unique patterns of results from each mixed effect model and as revealed by the Brinley plots. The reason for the directional inconsistency in a portion of the results between PWA and LHD groups was dependent on the inclusion of more cognitively demanding tasks that require complex cognitive computations. However, it is clear that the PWA group performed similarly to the LHD group on the sensory-motor tasks, but significantly slower than the LHD group when the cognitive demands increased independent of whether they were within the linguistic or nonlinguistic domain.

This conclusion is depicted in the theoretical schema presented in the Figure 5 (see Figure 11). According to this schematic, the difference between controlled processing A and B is dependent on whether more complex computations are required or not. The early processing stage requires relatively simple cognitive computation such as same or different discrimination. Based on the stages in the schema, the difference between the PWA and the LHD groups begins or exists even at the early processing stage (#3 in Figure 11) before the controlled processing A and B (#4 in Figure 11), which involves short-term and working memory. This finding indicates that the observed slowed language processing for the PWA group, on the complex language tasks such as sentence processing and lexical priming (Love et al, 2008) might be the product or outcome of slowed processing at earlier stages. Considering that the slowing occurred both in the PWA and the LHD groups, it's uncertain whether the aphasics' slowed performance in

complex language processing tasks is attributable to their aphasia or to the left-hemisphere damage, which they also have.

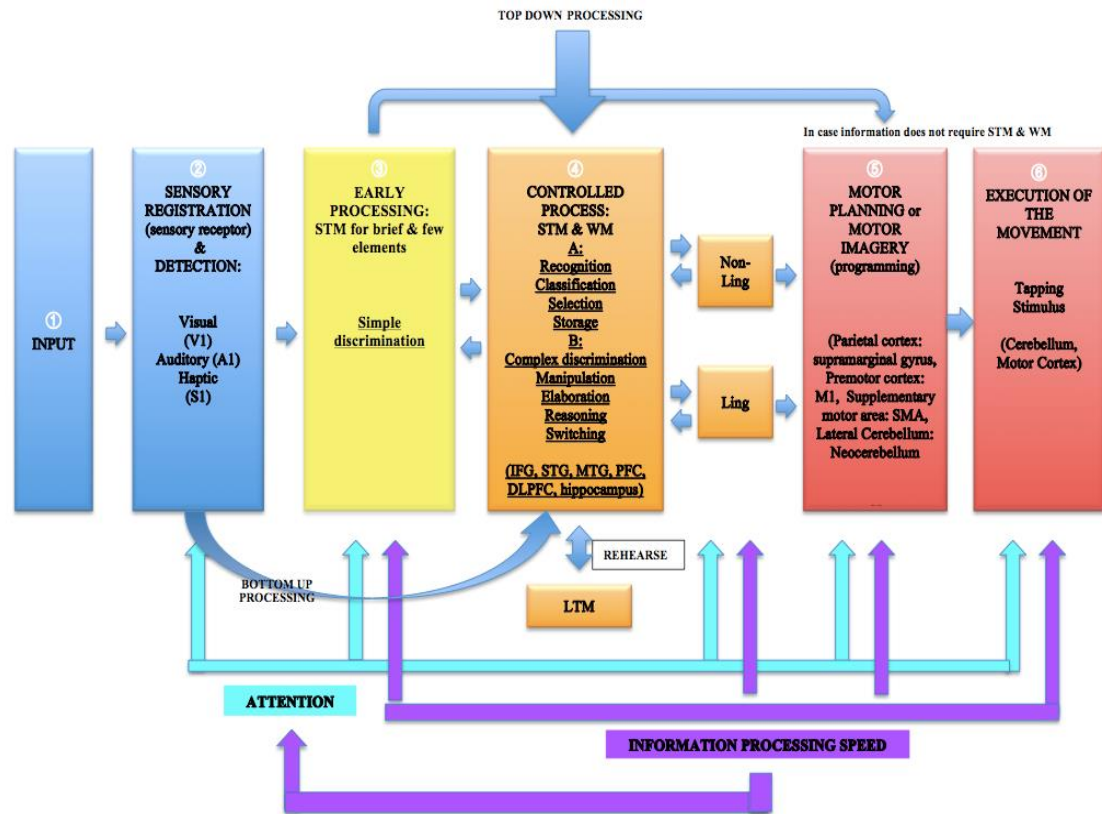


Figure 11. A schematic of information processing in PWA (revision of Figure 5)

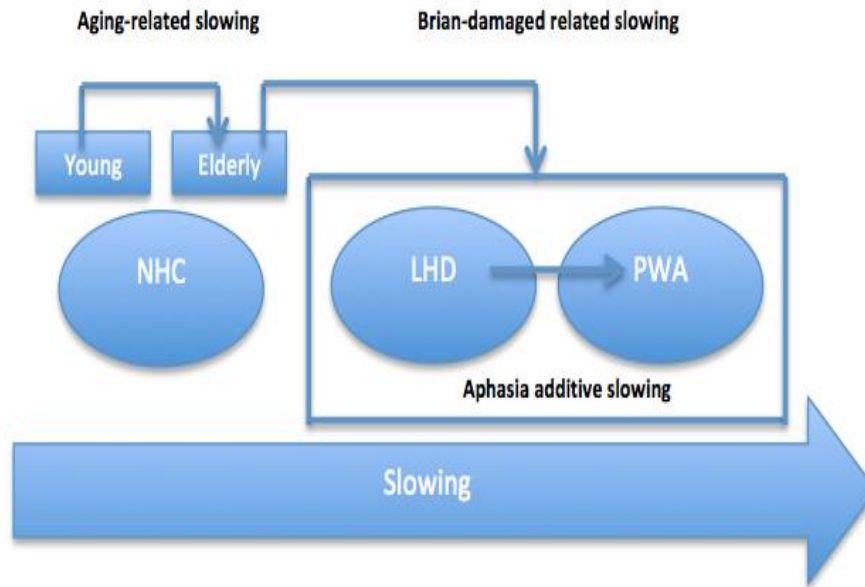
The two brain-damaged groups performed significantly and consistently slower than the NHC group regardless of the level of cognitive task complexity. Therefore, the slowing that is shared between two pathologic groups and that separates them from the NHC group is identified as brain-damaged related slowing. However, the two brain-damaged groups performed similarly to each other at the level of perceptual-motor (CRTT-RT1-3) tasks, and the two groups were different from each other for all the rest of the tasks from the perceptual to the higher-level cognitive tasks. The evidence for aphasia-specific slowing, derived from differences between

the PWA and the LHD groups was evident at relatively low levels of cognitive processing, such as making same or different decisions, through more demanding tasks such as those required for completion of the RCPM and sentence completion tasks.

The answer to the two primary experimental questions posed is as follows: Aphasia specific slowing exists, but left-hemisphere damage-related slowing also exists. Therefore, slowing associated with aphasia might be due to both the aphasia specific slowing and the brain-damaged related slowing. Domain specificity was not found, namely slowing occurred domain generally in both linguistic and nonlinguistic tasks in the PWA.

This study possesses several limitations. First, since no previous study investigated a LHD group without aphasia, this study has no direct comparisons. Studies investigating LHD people without aphasia, and comparing them with PWA participants should be conducted to replicate the findings of this investigation and to determine if this group represents a continuum of language impairments from normal non-brain-damage to aphasia (McNeil, 1982; 1988; also referred to as the “continuity hypothesis”; Freud, 1953, Chapman & Chapman, 1980, Buckingham, 1999; 2006). Considering that the LHD group exhibited different degrees of slowing performance compared to the participants with aphasia, not a difference in pattern of impairment, these data are consistent with this hypothesis. From this study, the LHD group might be located in between the PWA and the NHC groups. Therefore, the continuum can be the NHC-to-LHD-to-PWA continuum (see Figure 12) based on this study.

Care interpreting the results from the PWA group is warranted because the results are consistent with a left hemisphere lesion instead of the presence of aphasia per se.



**Figure 12. Normal-LHD-to PWA continuum**

As a future study, the functional connectivity of brain networks, in the resting state, should be compared between the LHD without aphasia and PWA group. It will be important to investigate whether the LHD group exhibits hypo-connectivity that is in some way comparable to that of the PWA group. Critically, the language performance of the LHD group must be assessed using cognitive/language tasks that are more demanding than those employed in the current study and thus likely to reveal subtle deficits if present. This would be necessary to correlate with impairments of connectivity in order to shed light on the normal-to-aphasic continuum and to further test the aphasia specificity hypothesis.

Additionally, future studies should employ different kinds of mental processing tasks to determine whether there are differential aspects or impacts on the degree of slowing depending on the nature of information engaged, such as syntax and phonology. Testing a complexity effect based on the dataset from numerous tasks would be necessary to confirm whether the

difference in degree of slowing is derived from the degree of cognitive task complexity. In the current study, the two most complex tasks (RCPM and SC), as indexed by response time, are too limited to determine whether the degree of slowing differs depending on the level of task complexity.

## APPENDIX A

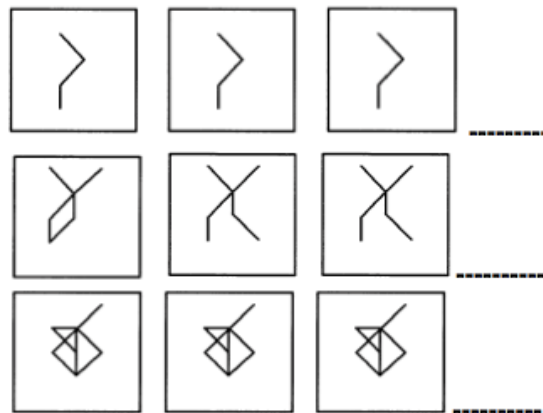
### NON-LINGUISTIC STIMULI: PATTERN COMPARISON

#### Pattern Comparison

In this test you will be asked to determine whether two patterns of lines are the same or different. If the two patterns are the SAME, press the number 1 key button.

If they are DIFFERENT, press the number 2 key button. Please try to work as rapidly as you can, answering to each pair of line patterns.

Try the following examples.





## APPENDIX B

### LINGUISTIC STIMULI: LETTER COMPARISON

ID # \_\_\_\_\_

#### Letter Comparison

In this test you will be asked to determine whether two strings of letters are the same or different. If the letters in the two strings are the SAME, press the number 1 key button. If they are DIFFERENT, press the number 2 key button. Please try to work as rapidly as you can, given an answer for each letter string. Try the following examples:

X \_\_\_\_\_ M

P \_\_\_\_\_ P

B \_\_\_\_\_ L

## APPENDIX C

### LINGUISTIC STIMULI: LINGUISTIC STIMULI: LEXICAL LISTS

Instruction: In this test, you will see either a word or nonword on the computer screen. Then you will be asked to determine whether the item is word or nonword with pressing Yes (Y: word), or No (N: nonword) buttons.

EASY 36 items	WORD 18 items	twisty, toxin, taxis, smoky, placid, hermit, frosty, fleshy, fondly, fizzle, evoked, detest, camels, canoes, bossy, bonnet, arouse, adored
	NON- WORD 18 items	afoub, aigy, boltem, bualt, huzzer, galiph, parver, tavort, ponsul, fonvex, dentaw, friven, infoct, slorid, fluko, gashis, gurrah, lorynx
HARD 36 items	WORD 18 items	wryly, yeasty, turbid, tartly, stamen, snivel, sextet, resole, reflux, purism, pestle, outran, nympho, nobler, muslin, mutton, levies, lapels,
	NON- WORD 18 items	agazed, advise, brusky, pausal, corpis, defirm, femar, gamit, baggot, lucous, pelvac, behash, salva, sulfar, drauma, usser, uglid, witsy

## APPENDIX D

### SELF-REPORT HISTORY FORM (Only for NHC group)

Have you ever experienced difficulties or been diagnosed with?

1. brain injury or disease? Y/N

If YES, please describe it. \_\_\_\_\_

2. a movement disorder such as paralysis or Parkinson disease? Y/N

If YES, please describe it. \_\_\_\_\_

3. a psychological disorder such as depression or bipolar? Y/N disorder

If YES, please describe it. \_\_\_\_\_

4. a visual problem even after vision correction (including eye glasses and eye surgeries)? Y/N

If YES, please describe it. \_\_\_\_\_

5. speech or language problem such as aphasia? Y/N

If YES, please describe it. \_\_\_\_\_

6. dyslexia? Y/N

If YES, please describe it. \_\_\_\_\_

## APPENDIX E

### DEMOGRAPHIC INFORMATION & DESCRIPTIVE MEASURES FOR PWA GROUP

Group P=PWA	Age	Gender	Education	POT	Lesion Site	ABCD	SRP	ROCFT	Sentence Span	Subtract2 Span	Alphabet Span	STM: FW Span
<b>P1</b>	68	F	14	9y	Left CVA	95.24	106	31	2	3.5	2.5	5
<b>P2</b>	53	M	14	7y	Cerebral hemispheres: Periventricular encephalomalacia	77	100	33	4	3	2.5	4
<b>P3</b>	69	M	16	1y10m	Cerebral Artery Occlusion, unspecified with cerebral infarction	94.73	94	31	2.5	3	3.5	6

<b>P4</b>	69	M	14	4y	Left hemorrhagic cerebrovascular accident (CVA): Cerebral artery occlusion with cerebral infarction	81.8	141	34	2	4	3.5	6
<b>P5</b>	70	F	16	11y	Left middle cerebral artery cerebrovascular accident (CVA)	104.76	151	27	2.5	2	2.5	3
<b>P6</b>	84	M	16	4y	Description: Moderately large area of restricted diffusion is seen on the DWI images in L frontotemporal, temporal, subinsular and basal ganglion regions involving caudate & lentiform nuclei. No hemorrhagic transformation. Corresponding high FLAIR T2 signal intensity is noted. Some periventricular FLAIR hyperintensity may represent chronic ischemic changes. Some local mass effect on L sylvian fissure but there's no hydrocephalus or gross midline shift. Affected Lobes: Left temporal, left frontal, left subcortical	100	71	33	0	2	2	5
<b>P7</b>	53	F	16	10y7m	Left CVA Large area of restricted diffusion identified w/in L MCA destruction. Predominantly involve the posterior L frontal lobe, anterior left parietal lobe and L basal ganglia. There's relative sparing of the anterior L temporal lob Affected Lobes: left parietal, left frontal, left subcortical Affected Vasculature: left MC	166.67	11	31	0	0	2	2
<b>P8</b>	68	M	15	5y1m	L MCA infarct	100	0	34	0	0	0	2

<b>P9</b>	65	M	16	9y	Left frontal	123	61	30	0	0	2	2
<b>P10</b>	69	M	18	6y2m	Left hemorrhagic cerebrovascular accident: left MCA CVA hemorrhage into infarct	116.67	12	30	0	0	0	2
<b>P11</b>	59	F	16	6m	Description: Evolving infarction L basal ganglia, corona radiata, centrum, semiovale, middle frontal gyrus, amygdala. Focal infarction L parietal lobe. Edema w/increased mass effect on frontal horn of L lateral ventricle. No midline shift. Small foci of petechial hemorrhages w/out a large intraparenchymal hematoma Affected Lobes: left parietal, left frontal, left subcortical Affected Vasculature: left MCA	90	119	34	2	2	3	4
<b>P12</b>	48	F	16	5y2m	Description: There's a large area of diffusion restriction in the left parietal and temporal lobes consistent with acute infarct. There's no hemorrhagic conversion Affected Lobes: left temporal, left parietal Affected Vasculature: left MCA	104.55	137	34	3.5	4	4	5
<b>P13</b>	65	M	16	3y3m	Large L MCA stroke Left MCA	133.33	14	33	0	2.5	2	4
<b>P14</b>	65	M	14	7y1m	L MCA territory infarct Left MCA	100	15	35	0	0	0	2
<b>P15</b>	58	M	14	31y6m	Left frontoparietal region Affected Lobes: left frontal, left parietal	100	76	36	0	0	0	2
<b>Mean</b>	64.2		15.4			106.27	73.86	32.4	1.23	1.73	1.82	3.6

## APPENDIX F

### DEMOGRAPHIC INFORMATION & DESCRIPTIVE MEASURES FOR LHD GROUP

Group L=LHD	Age	Gender	Education	POT	Lesion Site	ABCD	SRP	ROCFT	Sentence Span	Subtract2 Span	Alphabet Span	STM: FW Span
<b>L1</b>	57	F	16	11y 5m	Encephalomalacia within the left MCA territory in the left frontal lobe Affected lobes: Left frontal Affected Vasculature: Left MCA	109.1	140	36	5	5	3	5
<b>L2</b>	76	M	15	2y 4m	Description: Left periventricular; posterior temporal; basal ganglia Affected Lobes: Left temporal, left subcortical	117.6	137	32	2.5	5.5	3.5	2.5
<b>L3</b>	80	M	14	1y 8m	Acute ischemic stroke Description: Restricted diffusion L insular/left basal ganglia/corona radiate plus rounded area of restricted diffusion in the left lateral aspect temporal lab. These findings are consistent with an acute infarct in the left MCA distribution. Other tiny punctate foci of restricted diffusion in the brain parenchyma probably procedure related. Affected Lobes: left temporal, left subcortical, left other Affected Vasculature: Left MCA	95.2	177	36	2	4.5	3.5	2
<b>L4</b>	49	F	14	11y	Acute ischemic stroke	95.83	185	31	3.5	6	4	3.5

					Description: infarct L basal ganglia Affected Lobes: Left subcortical							
<b>L5</b>	26	F	17	2y	Description: Scattered small foci w/in L MCA distribution, especially L precentral gyrus, are seen with restricted diffusion on D-W I, and abnormal T2 prolongation on T2 FLAIR concerning for subacute infarcts, likely associated with/L MCA distribution emboli. Surrounding small areas of localized edema around these infarcts are noted, without mass effect or middle shift. No evidence of hemorrhage on the blood sensitive sequences Affected lobes: left frontal, left other Affected vasculature: left MCA	84	212	36	6	5.5	5	6
<b>L6</b>	80	F	16	1y 8m	Acute ischemic stroke Description: There are several tiny acute foci of stroke in the distribution suggested by the CTA, in the posterior left MCA distribution. There's no evidence of hemorrhagic transformation Affected lobes: left other Affected Vasculature: left MCA	100	206	31	3.5	4	3.5	3.5
<b>L7</b>	61	M	15	12y 1m	Description: Ventricles, cortical sulci and basilar cisterns are normal. Restricted diffusion L frontoparietal cortical sulci expending into insular region consistent w/ acute infarct. Diffusion restriction in region of L caudate nucleus & L basal ganglia. Distribution of LMCA. No midline shift. No intracranial hemorrhage. Posterior fossa is	122	188	35	2	4	3.5	2



					unremarkable. No pathologic extra-axial collections. Affected Lobes: Left parietal, left frontal, left subcortical Affected Vasculature: left MCA							
<b>L8</b>	46	M	13	11m	Acute ischemic stroke Description: Subacute infarcts of L basal ganglion, L subsegmental frontal cortex, & L caudate, w/a small amount of petechial hemorrhagic component. Small amount of subarachnoid blood in L sylvian fissure Affected Lobes: left frontal, left subcortical, left other Affected Vasculature: left MCA	109.52	232	34	5	5	4	5
<b>L9</b>	53	M	12	3m	Description: Small focal areas of decreased diffusion identified within the left parietal occipital region including subcortical white matter and centrum semiovale corresponding to the left ACA/MCA watershed distribution Affected Lobes: left occipital, left parietal, left other Affected Vasculature: left ACA, left MCA	85	135	29	2.5	2.5	3	2.5
<b>L10</b>	69	F	20	3m	Description: Areas of infarction involving the left MCA territory, predominantly within the basal ganglia and caudate regions Affected Lobes: Left subcortical Affected Vasculature: Left MCA	91.67	213	33	4	4.5	4	4
<b>L11</b>	67	F	12	5y 2m	Acute ischemic stroke Description: Patchy areas of restricted diffusion w/in L cerebral hemisphere in parts of MCA territory including in L frontal & parietal lobes involving the basal ganglia in L peri-sylvian region	88.89	160	33	0	4.5	3.5	0

					consistent with area of recent infarction Affected Lobes: Left subcortical, left frontal, left parietal Affected vasculature: Left MCA							
<b>L12</b>	76	F	12	7y	Life middle cerebral artery (MCA) distruption	87.5	188	33	0	3.5	3	0
<b>L13</b>	59	M	15	3m	Acute ischemic stroke Description: Patchy areas of restricted diffusion w/areas of core infarction involving L posterior frontal periventricular white matter extending into L corona radiata, L insular cortex and portions of L frontal & temporal lobe bordering L sylvian fissure. Affected Lobes: Left temporal, left frontal, left others Affected vasculature: Left MCA	91.3	175	34	3.5	4	3.5	3.5
<b>L14</b>	60	F	12	16y	Left MCA territory: Multiple embolic occlusions in distal branches of ACA, MCA and PCA branches Description: L medial frontal, L posterior frontal, L parietal Affected Lobes: Left frontal Left parietal	104.34	172	34	2.5	4	3.5	2.5
<b>L15</b>	54	M	18	6y 8m	Description: Acute lacunar nonhemorrhagic infarct w/in posterior L parietal cortical region seen as a restriction defect on the diffusion & ADC mapping imaging. This measures 7.5mm. No other areas of signal abnormality are seen in the gray and white matter regions Affected Lobes: Left parietal	95.45	202	34	4	4	3.5	4
<b>Mean</b>	63.05		15.05			103.65	135.7	32.74	2.24	3.27	2.94	2.24

## APPENDIX G

### DEMOGRAPHIC INFORMATION & DESCRIPTIVE MEASURES FOR NHC GROUP

Group	Age	Gender	Education	ABCD	SRP	ROCFT	WM: Sentence Span	WM: Subtract2 Span	WM: Alphabet Span	STM: FW Span
1	82	F	18	100	174	36	4	7	4	7
2	59	F	18	100	247	36	4	3	4	6
3	72	M	16	104.2	247	36	0	4.5	4	5
4	59	F	18	100	216	36	4	4.5	5	7
5	67	F	17	100	208	32	3	4	4.5	7
6	61	F	13	100	207	32	2	4.5	4	6
7	60	F	18	104.35	267	36	6.5	6	5	7
8	69	F	18	95.45	217	35	4	5	4.5	7
9	62	F	13	87.5	219	31	4	3	3.5	5
10	59	F	12	100	89	33	2.5	5	3	6
11	67	F	18	100	241	33	0	5	3.5	6
12	70	F	18	100	210	33.5	4	4	4.5	6
13	65	M	16	95.83	217	34	0	4.5	3	6
14	57	F	14	95.45	141	32	4.5	5	3.5	5
15	76	M	16	100	231	36	4.5	4	4	7
16	57	F	22	95.83	229	36	3.5	4	4	5
17	60	F	16	100	233	36	4.5	4.5	4	6
18	76	F	16	96	249	32	3.5	4	3	5
19	55	F	16	100	291	35	2.5	5	5.5	7
20	59	F	13	100	211	31	4	4	4	7
21	58	F	16	104.16	243	36	4.5	6	4.5	7
22	62	F	18	108.7	266	36	5	6	5	7
23	69	F	18	91.67	231	36	5	5	5	7
24	64	F	20	104.16	213	34	4.5	7	5.5	7

<b>25</b>	65	F	15	108.7	273	36	4	6.5	5	7
<b>26</b>	65	M	16	91.67	269	35	2.5	3	4.5	7
<b>27</b>	62	F	16	91.67	219	36	5	6	4.5	7
<b>28</b>	71	M	18	133.33	195	35	4.5	5	4	7
<b>29</b>	62	F	18	95.65	219	36	4	5	4	7
<b>30</b>	62	M	19	100	271	35	4.5	5	4.5	7
<b>Mean</b>	64.4		16.67	100.14	224.77	34.55	3.62	4.83	4.23	6.43

## APPENDIX H

### CAT SCORES OF PWA

PWA	Listening	Speaking	Reading	Writing
1	122	254	122	90
2	126	231	118	94
3	112	155	91	86
4	127	235	126	90
5	119	216	113	64
6	119	214	110	63
7	88	84	57	58
8	36	0	46	22
9	109	198	93	51
10	108	157	107	16
11	126	212	120	88
12	123	224	122	84
13	122	186	114	50
14	84	113	54	28
15	89	163	92	52
Mean	107.33	176.13	99	62.4

## APPENDIX I

### CAT SCORES OF LHD

LHD	Listening	Speaking	Reading	Writing
1	136	256	132	104
2	136	267	132	116
3	136	282	132	102
4	136	278.5	132	114
5	136	289	132	109
6	138	299	132	109
7	132	247	126	98
8	140	270	132	121
9	136	275	132	91
10	138	284	132	135
11	134	279	132	102
12	134	271	132	102
13	136	277	132	100
14	140	290	132	104
15	140	293	130	110
Mean	136.53	277.17	131.47	107.8

# APPENDIX J

## INDIVIDUAL RAW RT

Sub ject	RT 1	R T 1 - S D	RT 2	RT 2- SD	RT3	RT3 -SD	PA1	PA1 -SD	PA2	PA2 -SD	RCP M	RCP M- SD	LE1	LE1 -SD	LE2	LE2 -SD	LX 1	LX 1- SD	LX 2	LX- SD	SC	SC- SD
1	298.63	26.43	532.00	112.17	229.46	605.46	250.14	839.44	565.44	281.27	1388.35	9425.83	141.52	354.54	326.94	854.21	185.50	759.34	260.12	136.25	1256.89	1279.88
2	192.65	10.05	323.21	48.53	122.00	212.00	187.20	652.32	374.37	166.44	6438.53	3126.44	903.86	200.49	210.55	809.17	126.65	177.41	154.58	620.58	8927.90	7406.49
3	220.81	25.37	399.28	121.65	220.83	878.36	403.26	236.98	550.71	314.43	8609.58	4107.68	124.85	190.33	233.74	532.33	163.22	662.95	228.78	100.54	7925.31	5203.68
4	328.31	437.84	200.403	266.793	438.144	8384.33	2551.27	120.519	245.31	255.139	104.237	133.94	490.015	269.92	981.1205	9537.94						
5	329.75	6.21	428.52	106.96	221.448	324.627	165.043	717.228	266.149	1039.403	5478.84	106.764	231.97	253.349	683.08	174.918	522.77	279.760	122.160	1169.300	8819.01	
6	266.01	79.85	397.62	90.34	280.617	103.497	265.078	129.019	504.374	286.179	1039.403	5478.84	106.764	231.97	253.349	683.08	174.918	522.77	279.760	122.160	1169.300	8819.01
7	246.14	9.36	406.93	97.15	195.200	386.92	138.200	372.993	351.636	195.607	6320.09	2563.09	759.18	125.95	129.20	220.435	108.91	362.156	136.056	404.26	1195.695	9145.10
8	233.13	13.94	328.41	49.81	217.400	637.400	252.427	162.623	507.183	260.338	8091.64	3268.46	104.68	801.895	196.00	710.497	119.30	436.845	194.456	106.12	3898.12	4676.62
9	291.37	668.284	155.306	235.111	435.94	8876.50	3793.78	966.265	257.178	850.122	470.397	141.510	670.77	1919.1439								
10	254.97	580.97	276.607	456.179	776.293	1268.3177	206.418	793.183	219.395	345.118	2024.8782											
11	196.65	339.57	198.107	295.133	506.250	811.3533	963.160	189.589	119.237	135.419	1155.8866											
12	245.22	311.83	144.454	135.430	278.127	5392.3570	595.121	104.248	815.177	166.910	8683.9144											
13	291.37	476.347	667.352	333.123	410.136	1362.5585	133.454	195.346	163.535	371.97	1719.1715											
14	392.93	532.168	248.724	263.938	465.231	1188.6693	130.130	277.782	171.628	214.671	8502.3817											
15	273.84	398.62	362.171	299.114	506.289	1114.6238	160.400	293.733	214.866	368.140	2968.2956											
16	323.17	510.349	17.363	236.915	547.250	8908.6061	104.255	224.933	104.194	186.110	1173.1175											
17	198.88	539.144	207.600	172.573	207.575	7969.3777	729.157	145.558	755.165	110.455	7508.5540											
18	348.45	602.340	235.502	204.521	304.101	8423.3504	133.235	216.556	159.430	195.533	9128.6532											
19	234.37	303.39	147.331	103.283	175.639	5154.2169	566.118	910.169	613.95	112.355	5088.4176											
20	348.45	371.108	148.403	153.626	343.177	4957.2998	652.158	115.274	671.93	944.350	6513.6513											
21	241.38	357.32	238.879	212.999	349.310	9041.4335	753.127	167.577	852.289	120.451	8737.7570											
22	227.51	382.66	198.582	230.103	395.235	8746.6148	930.249	172.749	124.710	233.142	1050.8235											
23	204.41	406.118	159.485	128.388	273.957	4363.1807	931.278	138.266	123.271	170.780	6877.5057											
24	291.12	437.159	287.977	320.158	468.226	7710.3705	125.462	332.790	168.481	274.123	1431.1356											
25	244.60	16.31	500.41	209.99	542.04	215.04	996.028	404.74	201.650	140.140	7910.32	3999.22	101.990	367.32	169.783	394.33	110.925	374.85	165.700	1039.8951		
26	248.13	287.39	169.605	246.68	882.574	252.1003	5896.87	100.255	218.738	667.131	593.272	955.7383	5368.57									
27	327.66	385.76	607.241	257.144	485.990	490.900	683.742	0.353	87.400	106.295	187.696	113.342	129.911	434.1164	1032.086							
28	350.90	321.60	109.915	180.492	355.144	6061.56	2575.69	957.48	216.19	169.412	438.04	120.074	598.48	208.329	840.56	1183.341	1195.129					
29	307.24	359.47	163.632	250.110	545.239	1452.1127	105.174	167.402	158.546	361.254	1249.1626											
30	222.00	219.155	118.167	148.259	0.415	259.04	6.15	9.77	34.60	95.11	803.75	58.00	9.50	10.63	74.74							
31	252.37	419.154	205.771	143.335	349.169	7991.3514	771.165	163.397	116.374	154.549	7425.4864											
32	219.74	360.77	207.874	133.420	319.158	9392.58	802.693	141.126	322.674	115.126	419.7534	8347.57										
33	237.22	274.53	197.720	135.510	394.198	6163.87	3639.827	172.121	239.792	148.119	333.6119	5566.99										
34	240.17	410.39	207.133	165.658	275.118	6486.58	3227.776	133.125	219.933	159.138	406.6508	2647.100										
35	219.11	338.65	173.600	149.608	369.208	5857.3417	807.174	115.305	703.118	133.795	5159.3887											
36	272.44	339.81	153.153	131.332	205.780	5717.2783	820.186	117.355	875.288	106.427	6165.3926											
37	214.52	300.24	161.855	157.665	314.135	5811.3150	659.876	115.238	650.97	107.304	4144.3060											
38	300.10	439.56	206.840	130.209	332.117	5471.1742	880.116	149.302	941.156	141.494	5090.3773											
39	287.12	70.00	386.65	273.203	152.307	199.765	4490.1627	993.212	174.460	104.210	145.532	5323.3880										

40	198	5.9	278	24.	173	334.	108	315.	269	116	6091	2983	647.	132.	152	473.	767.	223	114	475.	6397	5096
	.80	8	.93	83	5.93	49	4.82	25	7.14	4.73	.75	.80	95	70	7.18	80	63	.59	6.05	56	.57	.07
41	276	26.	434	70.	235	687.	186	614.	390	204	7101	3282	718.	107.	161	457.	764.	151	120	279.	7402	6477
	.94	53	.00	00	9.28	03	8.12	51	5.74	4.80	.50	.77	45	22	9.02	17	19	.52	9.55	38	.42	.43
42	213	9.0	305	47.	214	100	178	552.	272	106	8048	3644	760.	152.	119	260.	749.	125	162	667.	6694	6062
	.02	7	.03	11	9.76	4.07	1.10	02	1.00	0.83	.21	.30	51	01	8.88	03	97	.58	7.74	59	.38	.09
43	268	35.	427	64.	186	323.	200	914.	135	755	5959	2214	845.	163.	118	287.	113	345	127	361.	7714	5344
	.62	27	.31	18	0.10	87	3.90	88	5.82	8.00	.59	.30	05	94	2.60	24	4.66	.54	7.66	78	.61	.58
44	229	10.	342	49.	170	398.	166	961.	180	955.	5593	3231	713.	191.	144	534.	721.	115	788.	220.	6015	5394
	.35	72	.21	20	0.55	53	3.28	92	6.19	39	.28	.78	33	99	6.17	11	55	.60	78	39	.75	.82
45	248	7.2	377	161	162	270.	170	643.	302	125	7398	3282	922.	274.	145	416.	960.	267	185	110	6039	4663
	.07	2	.59	.11	5.93	51	5.02	33	1.62	7.75	.75	.83	14	70	9.05	28	06	.89	4.35	0.22	.85	.17
46	208	8.7	348	49.	155	401.	139	313.	289	109	5448	3455	646.	97.5	118	226.	785.	201	133	472.	6888	5849
	.57	0	.10	73	7.00	42	1.25	13	0.98	1.74	.76	.25	98	4	2.90	38	73	.07	4.41	30	.61	.63
47	223	5.7	359	33.	186	744.	171	591.	283	904.	5159	1889	789.	166.	164	440.	903.	147	166	531.	4742	3636
	.98	1	.07	51	2.38	63	6.40	90	0.05	90	.66	.09	30	77	1.45	24	32	.44	4.23	96	.88	.26
48	234	3.2	369	114	296	123	148	391.	205	546.	5148	2159	739.	115.	123	217.	805.	151	133	450.	6042	4325
	.40	0	.93	.34	7.03	7.86	9.49	64	5.00	65	.97	.96	69	07	1.55	39	80	.88	1.04	55	.58	.93
49	238	0.0	350	34.	140	386.	181	605.	312	109	5729	2595	683.	120.	106	266.	772.	134	118	331.	6222	5108
	.10	0	.93	87	6.00	00	6.46	20	4.81	7.12	.49	.53	97	55	4.85	47	97	.27	1.17	59	.68	.98
50	202	2.3	400	56.	178	565.	172	552.	324	178	7471	3491	875.	192.	123	337.	982.	259	146	624.	4289	3304
	.72	6	.07	53	8.00	00	2.08	68	4.82	5.21	.20	.67	62	84	7.31	09	77	.28	1.48	19	.45	.04
51	244	11.	307	30.	132	293.	132	474.	260	130	5571	2435	556.	59.2	107	130.	688.	116	116	391.	7100	6946
	.29	93	.55	57	3.00	00	0.45	24	0.93	0.32	.19	.20	45	1	7.67	70	91	.84	5.68	26	.82	.15
52	204	16.	413	80.	106	101	198	531.	314	129	4420	1448	992.	138.	145	240.	102	101	183	814.	6415	4771
	.99	79	.90	41	7.04	0.00	7.26	85	8.12	8.37	.53	.54	20	82	2.24	77	5.56	.86	8.77	74	.94	.22
53	252	15.	314	41.	151	368.	194	631.	401	165	7845	2978	707.	169.	181	586.	908.	266	239	178	7868	7049
	.69	22	.28	10	1.00	00	5.05	48	7.00	6.22	.52	.11	05	84	7.61	48	65	.15	6.15	6.74	.00	.77
54	247	35.	315	39.	140	325.	141	447.	284	120	5056	1271	622.	86.0	115	353.	690.	103	122	335.	4904	3072
	.07	03	.83	18	1.00	00	0.68	02	6.61	7.57	.23	.71	5	0.93	64	48	.80	1.83	46	.62	.14	
55	244	15.	349	127	141	524.	156	669.	287	138	4629	2080	709.	129.	922.	139.	762.	168	166	723.	5888	4313
	.56	31	.52	.29	5.88	65	3.70	44	3.23	2.65	.73	.39	57	81	55	04	83	.66	1.38	36	.15	.88
56	222	9.9	328	56.	160	367.	191	569.	435	225	4274	1771	844.	200.	163	559.	124	502	183	847.	5024	3290
	.52	0	.00	00	1.00	00	1.17	64	0.59	3.86	.03	.99	76	46	8.10	89	2.91	.35	4.69	65	.82	.60
57	218	22.	323	52.	156	331.	209	102	327	179	6200	3954	694.	81.8	145	350.	965.	144	170	863.	6407	4633
	.89	58	.21	33	6.28	87	1.67	3.82	8.14	5.11	.74	.48	41	3	0.41	27	53	.35	0.82	39	.66	.25
58	167	10.	350	202	151	405.	115	212.	164	452.	6105	2408	648.	70.2	111	203.	649.	56.	107	278.	4769	3506
	.08	29	.38	.12	5.00	00	1.92	34	9.54	25	.81	.36	32	7	3.56	88	15	62	3.45	20	.47	.65
59	238	5.6	347	71.	155	315.	138	523.	216	887.	4779	1868	624.	89.0	113	291.	779.	219	134	550.	5670	4176
	.19	7	.83	46	4.62	39	2.74	20	8.33	35	.68	.48	20	5	4.29	52	53	.98	5.12	55	.15	.14
60	229	3.0	332	66.	146	304.	182	795.	296	137	5384	2326	661.	97.1	106	163.	752.	136	989.	305.	4726	3091
	.03	5	.14	48	8.00	00	2.64	72	6.00	7.82	.88	.63	10	7	4.35	15	44	.16	62	43	.88	.22



## APPENDIX K

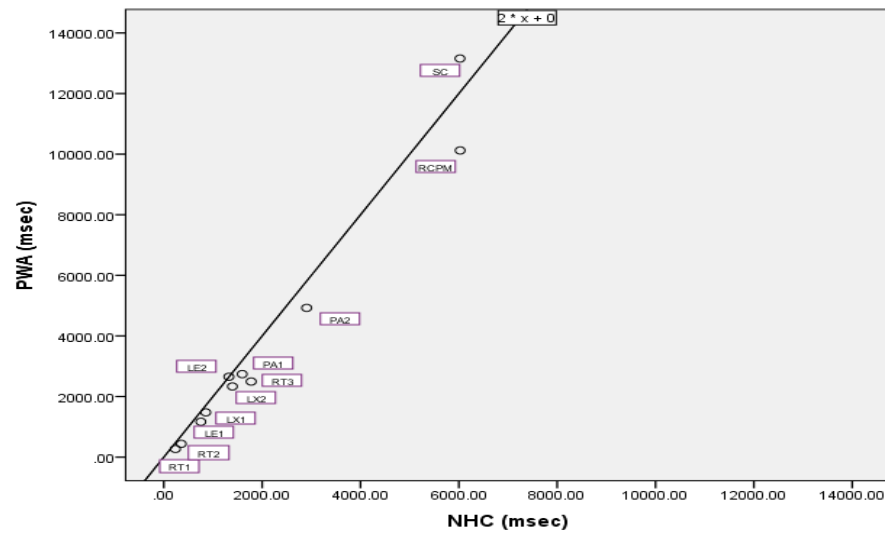
### LINGUISTIC STIMULI: ACCURACY (%)

Subject	RT1	RT2	RT3	PA1	PA2	RCPM	LE1	LE2	LX1	LX2	SC
1	100	100	100	95.45	91	63.89	95.35	93.02	100	66.7	88.23
2	100	100	100	100	93.18	91.67	97.67	95.35	100	80.56	91.18
3	100	100	100	97.73	93.18	94.44	100	95.35	100	75	79.41
4	100	100	100	93.18	91	86.11	100	95.35	100	61.11	79.41
5	100	100	100	95.45	88.64	86.11	100	90.7	100	66.67	91.18
6	100	100	100	97.73	88.64	88.89	100	97.67	97.22	69.44	67.65
7	100	100	100	100	91	88.89	97.67	90.7	94.44	52.78	67.65
8	100	100	100	100	93.18	72.22	86.04	97.67	88.37	55.56	51.51
9	100	100	100	95.45	81.82	72.22	97.67	93.02	88.89	58.33	76.47
10	100	100	100	97.73	86.36	52.78	100	95.35	94.44	52.78	52.94
11	100	100	100	95.45	91	88.89	100	93.02	97.22	61.11	79.41
12	100	100	100	93.18	91	97.22	100	97.67	100	80.56	100
13	100	100	100	95.45	81.82	86.11	100	90.7	97.22	69.44	64.7
14	100	100	100	97.73	61.36	61.11	100	88.37	88.89	52.78	20.5
15	100	100	100	97.73	93.18	75	97.67	97.67	91.67	55.56	67.65
16	100	100	100	100	93.18	94.44	97.67	100	97.22	88.89	97.06
17	100	100	100	88.64	77.27	63.89	95.35	86.04	94.44	69.44	91.18
18	100	100	100	97.73	91	80.56	100	95.35	91.67	72.22	94.12
19	100	100	100	91	95.45	94.44	100	97.67	100	66.7	94.12
20	100	100	100	97.73	91	100	95.35	95.35	100	58.33	94.12
21	100	100	100	97.73	79.55	75	100	97.67	100	88.89	97.06
22	100	100	100	95.45	88.89	72.22	100	90.7	100	63.89	82.35
23	100	100	100	97.73	91	94.44	95.35	95.35	97.22	69.44	88.23
24	100	100	100	84.1	72.72	44.44	95.35	97.67	100	63.89	88.23
25	100	100	100	97.73	95.45	86.11	97.67	95.35	97.22	83.33	94.12
26	100	100	100	100	95.45	86.11	100	97.67	100	61.11	79.41
27	100	100	100	88.64	91	66.67	97.67	97.67	100	80.56	91.18

28	100	100	100	95.45	95.45	97.22	97.67	95.35	100	58.33	85.3
29	100	100	100	97.73	91	88.89	100	95.35	94.44	72.22	88.23
30	100	100	100	97.73	93.18	91.67	100	90.7	97.22	61.11	97.06
31	100	100	100	95.45	95.45	97.22	100	88.37	97.22	94.44	97.06
32	100	100	100	93.18	95.45	88.89	97.67	97.67	100	58.33	94.12
33	100	100	100	86.36	88.64	86.11	100	95.35	97.22	77.78	94.12
34	100	100	100	95.45	93.18	100	100	97.67	100	77.78	100
35	100	100	100	93.18	93.18	94.44	100	93.02	100	88.89	94.12
36	100	100	100	88.64	81.82	88.89	97.67	95.35	100	55.56	82.35
37	100	100	100	97.73	95.45	97.22	100	97.67	100	86.11	100
38	100	100	100	91	91	100	100	97.67	97.22	94.44	88.23
39	100	100	100	93.18	79.55	63.89	97.67	97.67	97.22	63.89	91.18
40	100	100	100	93.18	86.36	91.67	97.67	95.35	100	61.11	82.35
41	100	100	100	93.18	91	83.33	100	95.35	97.22	61.11	97.06
42	100	100	100	93.18	81.82	83.33	97.67	97.67	100	72.22	94.12
43	100	100	100	93.18	91	97.22	97.67	97.67	94.44	88.89	97.06
44	100	100	100	91	86.36	69.44	95.35	93.02	97.22	55.56	73.5
45	100	100	100	97.73	77.27	88.89	100	93.02	91.67	97.22	97.06
46	100	100	100	95.45	93.18	94.44	100	97.67	97.22	75	97.06
47	100	100	100	95.45	77.27	91.67	100	95.35	100	83.33	97.06
48	100	100	100	88.64	77.27	83.33	100	95.35	100	77.78	94.12
49	100	100	100	97.73	95.45	91.67	100	95.35	100	86.11	97.06
50	100	100	100	91	91	69.44	100	97.67	88.89	77.78	88.23
51	100	100	100	95.45	95.45	75	100	95.35	100	88.89	97.06
52	100	100	100	97.73	95.45	91.67	100	97.67	100	94.44	100
53	100	100	100	95.45	95.45	88.89	95.35	97.67	100	97.22	94.12
54	100	100	100	95.45	95.45	86.11	95.35	95.35	97.22	97.22	88.23
55	100	100	100	100	91	94.44	100	97.67	100	91.67	100
56	100	100	100	97.73	93.18	91.67	97.67	95.35	100	72.22	100
57	100	100	100	97.73	95.45	100	100	95.35	100	77.78	97.06
58	100	100	100	95.45	84.1	88.89	97.67	90.7	100	91.67	91.18
59	100	100	100	97.73	91	91.67	100	95.35	100	69.44	97.06
60	100	100	100	100	95.45	91.67	100	97.67	100	86.11	100

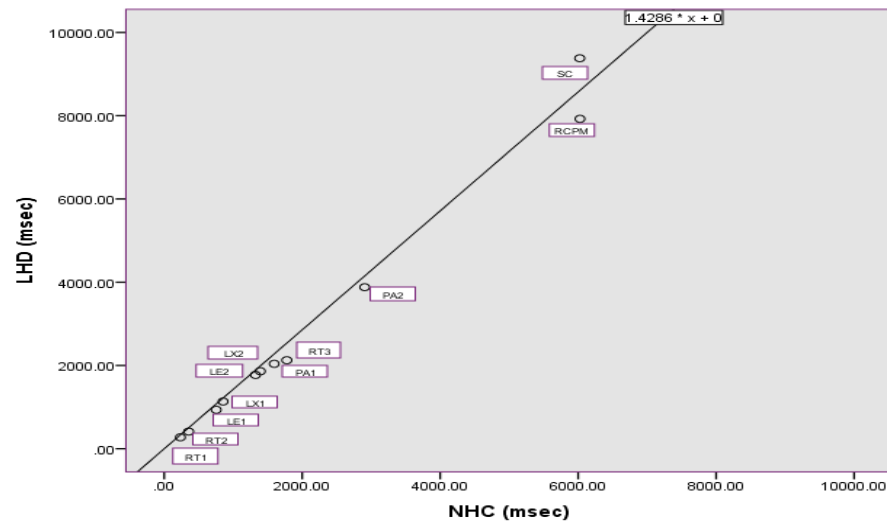
## APPENDIX L

### BRINLEY PLOT OF RT ACROSS 11 TASKS FOR PWA AND NHC



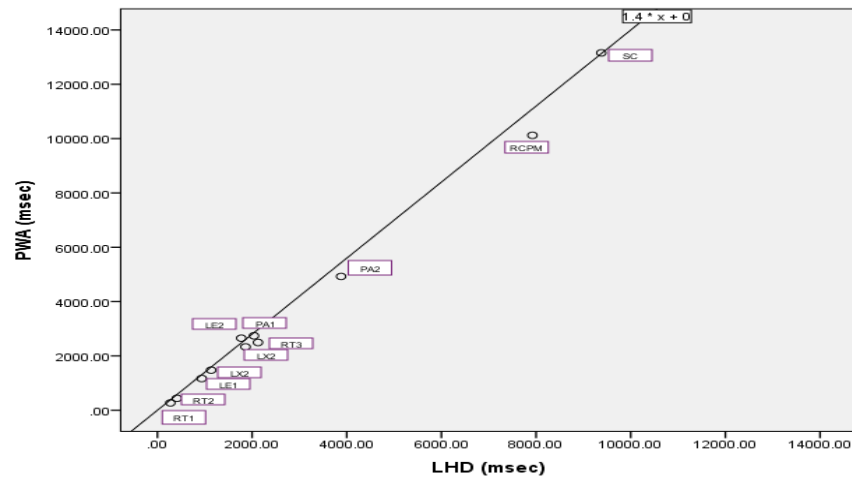
## APPENDIX M

### BRINLEY PLOT OF RT ACROSS 11 TASKS FOR PWA AND NHC



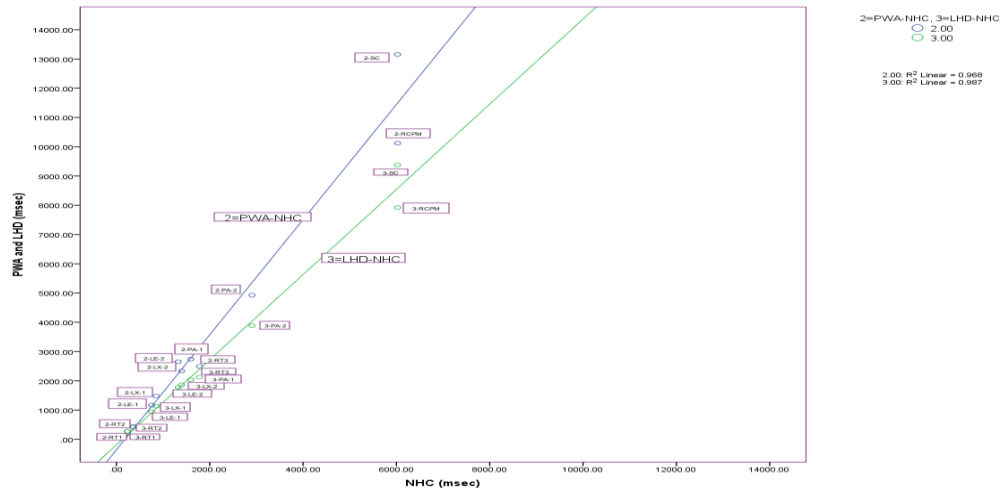
## APPENDIX N

### BRINLEY PLOT OF RT ACROSS 11 TASKS FOR PWA AND LHD



## APPENDIX O

### BRINLEY PLOTS OF RT BETWEEN PWA-NHC AND LHD-NHC FOR 11 TASKS DEPICTED IN APPENDIX L-O



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